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FATALITIES FOLLOWING OPERATIONS UPON THE NOSE AND THROAT NOT DEPENDENT UPON ANESTHESIA—A STUDY OF THREE HUNDRED AND THIRTY-TWO HITHERTO UNREPORTED CASES.*

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The development of the operative field of laryngology has been one of the most startling advances of medical science. From being an occasional operator, the laryngologist has become an accomplished surgeon with an enormous operative material—far beyond that of any other surgical specialist.

The work has become so attractive—or, perhaps, so remunerative—that there are today five thousand physicians in

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By resolution of the Society, the writer has been asked to continue and extend these studies. Accordingly, these cases will be included in those to be reported in the paper to be presented next year.

America, according to the American Medical Directory, who profess to be qualified to practice this specialty, one out of every thirty practitioners in this country.

In this large body all are not well trained, all are not skillful, all are not progressive, all are not mentally alert, all are not conscientious. Be this as it may, they have an important bearing on the well being of the people of today.

Considering the tremendous operative activity of laryngology, it is important to gain some estimate of its mortality risk. The fatalities must be excessively small in percentage, considering the number of operations. The meagerness of the literature on the subject shows this; and yet the published reports are sufficient to justify a more searching inquiry with the view of assembling reports of fatalities which have not been published.

It has been deemed advisable to exclude in this paper the anesthesia fatalities which would bear approximately the same relation to the total number of cases as in other operations of equal grade, with the slightly added danger which surgical work about the air passages would entail.

Inasmuch as the writer has no desire in this paper to touch upon cases in which the disease processes or the operative procedures were of themselves of a serious character, it was decided to limit the study to operations upon the nose and throat, as follows:

1. All intranasal operations on the walls of the nasal cavities and paranasal cavities, excluding pituitary operations and those performed for malignant disease, and
2. Tonsillectomy, adenoidectomy, incision for peritonsillar abscess. Endoscopic operations upon the larynx, trachea, bronchi and esophagus are excluded in view of the small proportion which the endoscopists bear to the total number of laryngologists. Furthermore, a study of such fatalities belongs more appropriately to an active endoscopist.

The writer became interested in the subject after the occurrence of a fatality in his practice following a submucous resection.

REPORT OF CASE.

H. C. C., an attorney, aged 52, consulted me on February 25, 1921, complaining of slight pain over the left ear for some

months, which had become somewhat worse lately, and which was more severe at night. His right ear had been deaf for a year, while in the left the hearing had just begun to diminish. Examination showed: Hearing: R., voice, 4 meters; whisper, 0.1; L., whisper, 4. Weber lateralized to the right. Rinne negative, right; positive, left. Nasal septum was greatly deflected to the right superiorly and to the left inferiorly. Diagnosis of chronic catarrhal otitis media was made.

Inflation improved his hearing very much and reduced the pain. It was felt wise to perform a submucous resection upon the septum in order to give him ample air for audition and for nasal respiration. Operation was accordingly performed on April 1, 1921.

He left the hospital feeling very comfortable on the next day. On April 4th he returned to the hospital on account of severe hemorrhage. The intern plugged his nose with gauze, which was allowed to remain until the following day when I removed it. In the afternoon he had a second severe hemorrhage, for which it was plugged again with gauze by the intern. This was removed that evening without a recurrence of any hemorrhage except a very slight oozing. On the day after his return to the hospital he developed acute tonsillitis on both sides. The patient was feeling very comfortable until three days later, when he was taken with pain in the occiput, marked deafness and an abscess in his left ear.

On the following day he was delirious, absolutely deaf, with an abscess in right ear but with no rigidity of neck. The head could then be rotated in all directions. No Kernig present. Examination showed bulging drum and hemorrhagic discharge from left ear. Paracentesis was performed, after which there was a copious purulent hemorrhagic discharge. During the evening manifest symptoms of meningitis appeared, and signs of facial paralysis, with neck rigidity and definite Kernig. He died that evening.

Autopsy showed middle ear on both sides filled with thin purulent material; purulent meningitis marked over the petrous bone on both sides; tuberculosis, left lung; pneumothorax, left side; arteriosclerosis; infectious thrombosis of liver; chronic interstitial nephritis.

There was no lesion over the cribriform plate on either side, and the sinuses were free from pus.

Only one other case falling within the scope of this paper came to the writer's observation through his practice.

This was a man whom I saw in consultation with a laryngologist who, in attempting to remove the superior turbinate bone, had fractured the cribriform plate. Meningitis developed within twenty-four hours and death ensued on the following day.

INVESTIGATION.

As has been already stated, the literature of the subject is exceedingly meager, being made up mainly of reports of isolated cases, with occasional autopsy protocols and with an attempt to explain the occurrence of death in the case reported. There has been very little constructive work done looking towards frequency of a fatal issue, and its reduction or elimination. The small number of cases reported militates against the expression of any conclusions which could be of much value.

The writer, therefore, undertook to assemble a larger number of cases, in the hope that a better understanding as to the frequency of such fatalities might be developed and that some conclusion of worth might be drawn. To this end a letter was sent to each of the five thousand practitioners of laryngology in this country, asking for reports of cases.

It is understood, of course, that such reports cannot have the weight of those which come from a single controlled clinical source. Factors of error are bound to creep in, depending upon the multiplicity of observers, the designed or unintentional coloring of the reports and much unavoidable looseness of observation. And yet there has been a surprising helpfulness manifested, which expresses the profound interest in the subject and the earnestness of the reporters themselves. Four hundred and sixty-seven answered that they had had no fatalities whatsoever. Two hundred and fifty made reports which this paper analyzes, and approximately 4,300 have thus far made no answer. As a result of this inquiry, I have reports of 332 fatalities, more or less complete and carefully collated

to avoid duplication. When this large number is compared to the paucity of published cases, it is obvious from the very first that we have not given to the subject the importance or the seriousness it merits, nor have we had sufficient data to justify conclusions respecting it.

It is, of course, impossible to determine with positiveness the causes of death in all the reports assembled, because there has been no set formula to guide these, and because of the variant conditions under which the observations have been made.

Altogether thirty-two autopsies are included, but these in many instances have been made by untrained men. The protocols are, therefore, not always of use. However, in spite of the possibility of inaccuracies as outlined, the writer feels certain that something of value can be had by the classification which has been made.

The following grouping of the fatalities is made with the number reported in each group:

I.	Meningitis	125
II.	Hemorrhage	55
III.	General sepsis	20
IV.	Erysipelas	8
V.	Endocranium (excluding meningitis)	26
VI.	Respiratory tract	43
VII.	Heart	5
VIII.	Miscellaneous	20
IX.	Undetermined	30
	Total	332

MENINGITIS.

Meningitis forms by far the most common cause of death. Although in the vast majority of the reports it followed nasal operations, in one instance it was the cause of death following adenoidectomy. The operation in this child, aged nine months, was performed to relieve convulsions, death occurring twenty-four hours later. It is likely either that the meningitis was present before the operation or that the endocranium was injured during the procedure.

There were four tonsil operations in this category. In considering tonsillectomy, no distinction is made as to whether or not there was a coincident adenoid operation. In two of these, an acute suppurative otitis media intervened between the operation and the meningitis, a possibility that can be readily understood. As is well known, a postoperative otitis media suppurativa is commonly of a virulent type.

In one of the other cases the meningitis followed immediately after operation; in the other, ten days later, both rapidly succumbing to the disease. The causal relations in these two cannot so readily be explained.

Meningitis was the cause of death after 120 nasal operations as follows:

1.	Intranasal frontal operation	16
2.	Probing and irrigating frontal sinus	7
3.	Ethmoid operation, with or without resection of the middle turbinate	39
4.	Sphenoid operation	10
5.	Maxillary operation	1
6.	Resection of middle turbinates	15
7.	Submucous resection	13
8.	Removal of polypi	13
9.	Combined sinus operation	1
10.	Combined submucous and sinus operation	5
	Total	120

This is certainly an appalling list, which cannot be lightly cast aside. The preponderance of fatalities is altogether on the side of the operations which come into relation with the lamina cribrosa. Perhaps the suggestion contained in the older books that the cribriform plate can be recognized by its hardness and resistance to the instruments may be somewhat responsible for the frequency of this fatal complication. In seventeen instances the reporters noted the occurrence of fracture into the cranial cavity, but there were doubtless many instances where this accident was overlooked.

Several reporters saw the cerebrospinal fluid escape during the operation. Another undertook to remove the superior

turbinate, without disturbing the middle turbinate. Another stated that he removed the entire cribriform plate which was sequestrated. Quite a few used the Ballenger ethmoid exenteration knife but discarded it after the fatality. In some, plugging the nose too long or too frequently appeared to be a factor. In others, an acute otitis media suppurativa incident to the operation was the forerunner, as in my own case.

In most instances, meningeal symptoms appeared within forty-eight hours after the operation. Can this be explained on the theory of an infection without fracture into the cranial cavity? If intracranial infection can make its presence known so soon after operation in the absence of fracture, what prevents its more frequent occurrence in the multitude of operations which disturb the bacterial relations? Are we wrong in asserting that the nasal cavities are most resistant to infections? Considerations such as these force us to the conclusion that most of the cases occurring within the first twenty-four hours are due either to a fracture or to the lighting up of a latent meningitis.

Of the nine reported autopsies made on patients dying from meningitis, fracture was found in five in whom the symptoms appeared during the first twenty-four hours; in one with no specification as to onset, and there was no fracture in the two cases sectioned in whom the symptoms appeared after forty-eight hours.

1. Intranasal Frontal Sinus Operation.—Considering the relative infrequency of the intranasal frontal sinus operation as compared with other intranasal operations, sixteen deaths from meningitis (13.3 per cent of all the cases) are sufficient to show the dangerous possibilities of this operation and to warn those whose knowledge of the anatomy is not sufficient to justify their undertaking it.

These considerations may not apply to the removal and curettement of the ethmoid cells, but they are forceful in reference to the drainage of the frontal sinus in connection with any attempt to enlarge its canal or orifice. One operator reports two cases in which the operation was followed by meningitis within twenty-four hours, one dying two days later and the other three days later. Another reports a case almost the counterpart of the first of these. In another, the menin-

gitis with unconsciousness supervened immediately after the operation with a temperature remaining above 105 until exitus three days later. In four, the symptoms appeared several days after the operation when the sinus condition had improved, justifying the opinion that there had been an infection without fracture. Another case is reported in which the operation was performed one week after the appearance of influenza, complicated by an acute frontal sinusitis. Two days later meningitis developed and the patient died three days later.

2. Probing and Irrigating the Frontal Sinus.—This operation which many consider entirely free from danger is responsible for seven deaths, large in proportion to the number of such procedures undertaken. One of them developed the disease in a few hours, two in twenty-four hours, one dying a day later, the other two days later. The disease appeared two days after operation in two of the cases, several weeks afterward in another, and in one case the symptoms may have appeared before the operation.

3. Ethmoid Operation With or Without Resection of the Middle Turbinate.—In more than one-third of the meningitis cases reported, this operation was specified as the cause. All types of operation are represented, and, according to the reports, it is impossible to determine which type is most dangerous, with the exception of the Ballenger knife, which appeared to be most culpable either by reason of its mechanical construction or from inexpertness in its employment.

As no operations in which ethmoid curettement or exenteration was combined with those upon the septum or other sinuses are included in this group, the ethmoid procedure may be accountable for more fatalities than are here stated.

In nine the meningeal symptoms appeared within a few hours; in ten others, within twenty-four hours; in five others, within forty-eight hours; in eight of the remaining they occurred later, while in seven the time was unspecified. The onset of the symptoms has an important bearing on determining whether fracture into the cranial cavity has resulted, for, as has already been suggested, it is hard to conceive of a meningitis of lymphogenous or hematogenous origin occurring within twenty-four hours after the infection pathway has been first opened.

Escape of cerebrospinal fluid at the time of operation occurred several times. One of these was followed immediately by meningitis; another, after two weeks. Packing for eight hours to control postoperative hemorrhage is reported to have been followed in forty-eight hours by a fatal meningitis. One of the patients was a baby two years old. In one instance the operator, a well known and most skillful laryngologist, after the onset of the meningitis, removed the cribriform plate in the hope of relieving the process. He found evidence of a chronic pachymeningitis.

4. Sphenoid Operation.—The sphenoid operation is looked upon by laryngologists as more hazardous than that upon any other sinus, and they have imparted this view in large measure to their patients; hence, laymen are more prepared for a fatal issue in this than in any other type of intranasal sinus operation. A more or less extensive ethmoid exenteration practically always constitutes a part of the sphenoid operation, and naturally either may serve as an etiologic factor of the meningitis. The reports do not differ materially from those upon the ethmoid operation. In one case the meningitis came on before night and death occurred within seventy hours.

5. Maxillary Operation.—Only one intranasal maxillary operation was followed by meningitis, following what appeared to be a simple procedure in a long standing process.

6. Resection of Middle Turbinate.—This has an unusually large toll—fifteen—for what is ordinarily considered a very simple affair. In only one was there a coincident operation performed, resection of the corresponding inferior turbinate. Three developed meningitis the first day, one died on the second day with symptoms of acute mania, and one followed middle ear abscess and meningitis, supervening upon the operation. The last was performed by a general practitioner. The reports of this group are very incomplete, consisting almost entirely of a statement of the operation and cause of death. In one, cutting adhesions between the turbinate and septum resulted in meningitis in twenty-four hours and death thirty-six hours later.

7. Submucous Resection of the Septum.—Two factors combine to influence a meningitis following submucous resection: The perpendicular plate of the ethmoid and the exposure of

the medial sides of the septal mucosa with exclusion of drainage brought about by holding the two exposed surfaces together. If there was much disposition to nasal infection in the nose, we should expect far more frequent expressions of that infection than we get from this operation. However, the fifteen cases here reported constitute a sufficiently large mortality rate, from meningitis at least, to be of much concern to operators. In most of the cases reported, in which the onset is specified, the meningeal symptoms did not appear until after the second day, substantiating the view that these cases are largely hematogenous or lymphogenous. In one, the symptoms developed the first day and the patient died on the next, the reporter being sure that a fracture was the cause. Autopsy revealed a puncture of the cribriform plate in another in which meningitis appeared soon after operation.

My own case, reported in this paper, comes under the category of secondary meningeal infection resulting from otitis media.

8. Removal of Polypi.—Although removal of polypi was one of the first nasal operative procedures to which our literature refers as a cause of meningitis and death, the thirteen cases here recorded will be considered a number far beyond the expectation of laryngologists. The natural inference would be that the forcible evulsion of the polypi with forceps must have caused a fracture into the cranial cavity or so close to it that infection was possible.

Several of the reports are exceedingly interesting. One patient became semistuporous within twenty-four hours. No autopsy was made but the operator thinks that a meningitis was developing at the time of the operation.

One surgeon removed a polyp from the superior meatus with a snare. He heard what he calls a rucking sound. Two days later the patient developed meningitis, which was soon followed by death. In another, an ethmoid exenteration had been done some time before; soon after the polypi were removed meningitis developed. The surgeon thinks that there was a necrosis of the cribriform plate which caused the acute meningitis by activation through the operative procedure. A patient immediately after the removal of several polypi from the nose fell into convulsions, and was taken with meningitis

followed by death. In another, from whom some polypi had been removed, autopsy showed polypi extending into the sphenoid. In another who died of meningitis eight days after operation, autopsy showed the lamina cribrosa on both sides as thin as paper through which the polypi in the ethmoid were visible. There was a very thin fracture running across the right cribriform plate and an abscess in the frontal lobe.

9. Combined Sinus Operation.—A combined sphenoid, maxillary and ethmoid operation resulted in meningitis and death in one instance.

10. Combined Submucous and Sinus Operation.—The operations performed were submucous and bilateral ethmoid exenteration, three, in one of which there was fracture of the cribriform plate with escape of cerebrospinal fluid; submucous, exenteration of ethmoid and intranasal operation on the frontal; and submucous resection with exenteration of ethmoid and intranasal operation.

HEMORRHAGE.

Hemorrhage constitutes the next most important group and is in evidence in both the nose and throat operations, as follows:

1. Tonsillectomy (with or without coincident adenoidectomy)	43
2. Adenoidectomy alone	2
3. Incision of peritonsillar abscess	3
4. Submucous resection	2
5. Sinus operation	4
6. Resection inferior turbinate	1
Total	56

1. Tonsillectomy.—It is certain that the forty-three deaths from hemorrhage after tonsillectomy do not by any means constitute all the unreported cases in this country. On the contrary, I am convinced that there are many more yet to be uncovered.

An analysis of the records shows that in sixteen there was a lack of postoperative control—that is to say, the operation was performed in the office and the patient sent home: or

the parents insisted on taking the patient away; or there was lack of observation whereby the bleeding might have been recognized; or the operator left the city; or the physician in charge was unfamiliar with methods of producing tonsil hemostasis. Most of these patients would have been spared under other circumstances.

Hemophilia, mainly unknown to the operator, is charged with seven of the fatalities; in one the operator discovered too late that the patient's brother had died from hemorrhage after tonsillectomy, the basis of which was hemophilia.

In another instance, familial hemophilia was discovered after the death of the patient.

A fatal result ensued in four cases in spite of thoroughgoing ligation of the vessels. The fatality was immediate in three cases. Persistent uncontrollable bleeding occurred twelve times and fatal secondary hemorrhage four times, one being after three weeks. In one of these cases the bleeding occurred five days after the operation, coming from the nasopharynx, the patient never having been seen by the operator after the operation.

There were two instances in which there was an anomalous branch of the carotid. In one this branch was found to leave the main trunk of the carotid at about the middle of the tonsil fossa, and the vessel was severed three inches from its origin. The section of the enucleated tonsil shows a portion of the arterial trunk of large caliber with two or three large branches, which supplied the upper portion of the capsule. Palpation of the tonsil, according to the operator, would have discovered this situation.

One patient was an idiot; another had extremely high blood pressure.

From these reports it is obvious that much can be done to reduce the mortality from tonsil hemorrhage.

2. Adenoidectomy.—Very little detail is given in these cases.

3. Incision of Peritonsillar Abscess.—Of the three cases, one was in a child in which the incision was followed by persistent bleeding, which continued for some days and for which treatment was of no avail. Of the second no detail is given. The third proved to be a hematóma, which had resulted from

erosion of the carotid or internal jugular, death being immediate.

4. Submucous Resection.—Two deaths from hemorrhage occurred after submucous resection; one was allowed to return home after three days, the physicians available not being expert enough to control it. As to the other, no details are given.

5. Sinus Operations.—Hemorrhage resulted in death following puncture of the maxillary sinus, the bleeding being from both the gums and nose. A resection of the nasomaxillary wall came to a fatal issue by reason of hemorrhage; another death after removal of polypi, and still another after ethmoid exenteration. In none of these cases was there anything to anticipate a fatal hemorrhage.

GENERAL SEPSIS.

The number of deaths from general sepsis is relatively small and is in keeping with the teaching that serious infections are rare in nose and throat operations, if we exclude meningitis. They include:

1. Intranasal maxillary operation	2
2. Submucous resection	1
3. Tonsillectomy	16
4. Incision peritonsillar abscess	1
Total	20

1. Intranasal Maxillary Operation.—In one of the cases symptoms of general sepsis developed three days after operation for an acute maxillary sinusitis, which resulted in death on the following day from general peritonitis, the appendix not being involved. In the second case, the death occurred two days after operation.

2. Submucous Resection.—In the single case reported, the fatal issue occurred eight days after operation. Packing had been retained for twenty-four hours.

3. Tonsillectomy.—There is a growing feeling that sepsis is more likely to occur under local anesthesia than under general. This is a measure confirmed by the reports which show that of those in which the anesthesia is specified, seven were local and two general. Localized swelling is characteristic of

the majority, tracheotomy, though without avail, being required in two. In one the pharyngeal tissues were found infected with colon bacillus; in another peritonsillar abscess developed; in another, chill, 106 temperature and abscess of the hip; another showed by cultures that there was an attenuated diphtheria infection.

4. Incision Peritonsillar Abscess.—It cannot be determined whether the general sepsis reported was due to the operation or to the process itself.

ERYSIPELAS.

The operator is accustomed to look upon erysipelas with more chagrin than any other complication unless it be preoperative. The grouping is as follows:

1. Submucous resection	4
2. Sinus operations	2
3. Resection middle turbinate	2
Total	8

1. Submucous Resection.—In one of the submucous cases the middle turbinate was also resected. One showed symptoms one day after operation, the others much later.

2. Sinus Operation.—Included in the sinus operations were one of removal of nasal polypi by snare, in which erysipelas developed on the second day, and one in which erysipelas was present at the time of the intranasal maxillary operation.

3. Resection of Middle Turbinate.—One of the erysipelas fatalities following resection of the middle turbinate was in an old man with a chronic nephritis; the other patient bled considerably, requiring a gauze plug which was not removed until the following day.

ENDOCRANIUM, EXCLUDING MENINGITIS.

This includes the following:

1. Brain abscess	5
2. Cavernous sinus thrombosis	9
3. Cerebral embolus or hemorrhage	12
Total	26

In the absence of autopsy the diagnosis of brain abscess and of cerebral embolus or hemorrhage must be more or less doubtful, but in the grouping the opinion of the operator is accepted.

1. Brain Abscess.—In one the diagnosis was confirmed by autopsy, in another by operation. In two, the operation performed was removal of polypi. One of these developed a frontal lobe abscess by extension from subperiosteal and subdural abscess. The other, two days after the operation, became drowsy with slight elevation of temperature and pulse rate from 50 to 70. He died in four weeks. One case resulted from retention of gauze after an ethmoid operation. Another had ear abscess a week after operation from which the brain abscess resulted.

2. Cavernous Sinus Thrombosis.—This extraordinary complication is probably due to a hematogenous infection which finds its counterpart in those cases of furunculosis and similar processes affecting the skin of the external nose, vestibule and lip. Altogether there were nine cases, divided as follows:

Tonsillectomy	3
Sinus operations	2
Incision peritonsillar abscess	1
Resection inferior turbinate	2
Series of nose operations	1

Two of the tonsillectomies were followed very soon by the symptoms, which developed very rapidly. The other was that of a young soldier who had had tonsillitis two weeks before. The operation was performed under local anesthesia without incident. On the fourth day he had a slight elevation of temperature, and on the following day there was beginning exophthalmos with slight blurring of the disc on the left side. Symptoms rapidly increased until exitus on the seventh day after the operation. Diagnosis was confirmed by autopsy. The sinus operations were puncture of the antrum followed by orbital cellulitis and exenteration of the ethmoid.

There was nothing extraordinary in the incision of the peritonsillar abscess, except the unusual character of the complication. In one of the cases of resection of inferior tur-

binate, an adenoidectomy was done at the same time; in the other, the operation was bilateral. In the last case, three or four unspecified nasal operations had been performed at intervals of two days.

3. Cerebral Embolus or Hemorrhage.—This complication is represented in the fatalities thirteen times:

Tonsillectomy	7
Incision peritonsillar abscess	3
Nose operations	3

Of the tonsillectomy cases, three were in patients over forty and appear to have been due to accompanying conditions. As to the other four, a reporter describes one as being some brain condition, probably thrombus; another is stated to have walked to the bathroom contrary to orders and succumbed after showing localized paralyses; another had hemiplegia from embolus of the right cerebral; the fourth died suddenly while sitting up in bed.

The three cases of incision of peritonsillar abscess are all reported by the same observer, who may have overlooked other possible causes.

The two nasal operations were upon patients fifty-six and fifty-eight years of age. One, upon whom a submucous resection was made, had been a hard drinker with a blood pressure of 230; the other died suddenly when he was put back to bed after an ethmoid exenteration.

RESPIRATORY TRACT.

The fatalities coming under this heading are classified as follows:

1. Foreign body	5
2. Edema larynx and pharynx	6
3. Pulmonary edema	1
4. Pneumonia	17
5. Pulmonary embolus and abscess	14
Total	43

The operations concerned with fatal complications involving the respiratory tract are as follows:

Nose operations	10
Tonsillectomy	31
Adenectomy	1
Incision peritonsillar abscess	1
Total	43

1. Foreign Body.—The five reports are as follows:

A submucous resection was performed under ether and the nose plugged with finger cot stuffed with gauze, which was inhaled and followed by death, the cause being unknown until autopsy showed the gauze stuffed finger cot in the primary bronchus.

A child died on the table after tonsillectomy from asphyxia due to clotted blood in the bronchi.

A very fat short necked colored woman died after incision of peritonsillar abscess from pus entering the larynx and causing suffocation.

A tooth was dislodged during tonsillectomy under ether, subsequently causing death from inflammatory reaction.

A boy before tonsillectomy under chloroform had eaten a large amount of beans. Immediately after the operation he vomited about a pint of beans, some of which were inhaled and caused death from suffocation.

2. Edema of the Larynx and Pharynx.—These cases may all have been septic, though some were exceedingly rapid. In one the edema was so rapid and severe that the patient died within twelve hours after the operation; another for whom tracheotomy was done within twelve hours survived two days. One died two weeks after operation, and two others in one week; still another died of edema glottidis one month after operation.

3. Pulmonary Edema.—This was found associated with a miliary tuberculosis on autopsy after a death from tonsillectomy.

4. Pneumonia.—When pneumonia follows an ether operation it is usually assumed that the anesthetic is responsible. In practically all of the cases here reported the anesthetic

etiology may be discarded. One was due to the inspiration of a piece of cartilage in a submucous resection under ether, causing foreign body pneumonia; two others were septic pneumonia from tonsillectomy, one under gas oxygen and one local; another after an intranasal antrum and frontal operation under local anesthesia developed septic pneumonia. The complication followed a curettement of the ethmoid exenteration and also an intranasal maxillary operation. Among the conditions blamed for pneumonia after tonsillectomy may be mentioned aspiration, influenza, recurring bronchopneumonia, a coincident peritonsillar abscess. All of these cases but one were under local anesthesia.

5. Pulmonary Embolus and Abscess.—Pulmonary abscess has been held as due to the general anesthesia, but the investigations of Cutler and Hunt (Arch. Int. Med., Chicago—1922—XXIX—449) show that it usually results from embolus. Hence it belongs under the category of this paper and not under that of anesthesia. Many more cases of pulmonary abscess will undoubtedly be discovered if the investigation is extended. All the cases of pulmonary abscess following tonsillectomy were general except one which was local and one not specified. In the reported exception the complication followed a resection of the inferior turbinate under general anesthesia. One reporter had three cases. Another gives the record of autopsy, which showed suppuration of the posterior ethmoid and sphenoid present. Of the four cases of pulmonary embolism, two were confirmed by autopsy.

HEART.

The five cases of heart cause of death comprised the following: Angina pectoris, after tonsillectomy; dilatation of the heart after removal of polypi; cardiac asthenia after tonsillectomy in a patient with grave heart lesions; cardiac infarct after incision of peritonsillar abscess and rupture of heart confirmed by autopsy in a submucous resection.

MISCELLANEOUS.

This list includes the following:

1. Acidosis	1
2. Delirium tremens	1
3. Dilatation of the stomach	1

4. Diphtheria	1
5. Epilepsy	1
6. Hyperthyroidism	2
7. Orbit injury	1
8. Poisoning	1
9. Scarlet fever	3
10. Shock	1
11. Status lymphaticus	5
12. Uremic coma	1
13. Uterine hemorrhage	1
Total	20

The acidosis fatality occurred in a diabetic upon whom a local tonsillectomy was performed.

The delirium tremens followed a sphenoid operation.

Acute dilatation of the stomach resulted, according to the reporter, from a heavy meal a half hour before an ethmoid exenteration.

Diphtheria caused the fatality after a tonsillectomy in a man fifty-five years of age.

The operator had no knowledge of previous occurrence of epileptic seizures in a patient who died in an attack after a resection of both turbinates.

Hyperthyroidism unrecognized previous to operation was held responsible for two deaths from tonsillectomy.

An inexpert operator pushed an instrument into the orbit in doing a submucous resection.

Another injected an overdose of adrenalin by mistake instead of novocain before a tonsillectomy, causing death.

Three cases developed scarlet fever after tonsillectomy and died.

Shock was held responsible for a fatal issue after tonsillectomy in a patient with goiter.

Four tonsillectomies and one ethmoid exenteration succumbed to status lymphaticus, in which, of course, the anesthetic relation must be considered.

Uremic coma caused the death of a man suffering from an intense nephritis upon whom a submucous resection, naso-

maxillary operation and an ethmoid exenteration had been done.

A woman with a high blood pressure expired suddenly from a profuse uterine hemorrhage sixteen hours after a tonsillectomy under general anesthesia.

UNDETERMINED.

In thirty cases no cause was determinable, either by the physician in charge or by the writer from the reports submitted. The operative procedures concerned were the following:

1. Tonsillectomy	16
2. Adenectomy alone	1
3. Exenteration ethmoid, with or without removal of polypi	4
4. Puncture of nasomaxillary wall, irrigation of the sinus	5
5. Submucous	1
6. Resection middle turbinate	2
7. Combined tonsillectomy and submucous resection	1
Total	30

The interesting cases in this group are those in which there was puncture of the nasomaxillary wall and irrigation of the sinus. They may well be compared with the sixteen already published and discussed at great length by Gording and Grove. There are doubtless some cases of status lymphaticus and unrecognized hemorrhage included in the group of undetermined causes.

TABULATION.

The table shows the cause of death in each type of operation. It is to be noted that tonsillectomy heads the list with 126, ethmoid exenteration being next with 46. The throat operations number 142, the nose operations 190.

TABLE SHOWING OPERATIONS AND CAUSES OF DEATH.

	Frontal	Ethmoid	Sphenoid	Maxillary	Resection Middle Turbinate	Resection Inferior Turbinate	Submucous Resection	Removal Polypi	Combined Sinus Operations	Combined Submucous and Sinus Operations	Combined Submucous and Resection of Either or Both Turbinates	Tonsillectomy	Adenoidectomy	Incision Peritonsillar Abscess	Total
Meningitis	23	35	14	1	15	....	13	13	1	5	....	4	1	....	125
Hemorrhage	....	1	....	2	....	1	2	1	....	....	....	43	2	3	55
General Sepsis	....	....	....	2	....	....	1	....	....	....	....	16	....	1	20
Erysipelas	....	....	....	1	2	....	2	1	....	....	2	....	....	....	8
Endocranium	....	4	....	1	....	2	1	2	....	10	1	....	1	4	26
Respiratory Tract	....	1	....	1	1	1	3	....	1	....	2	31	1	1	43
Heart	....	....	....	....	....	....	1	1	....	....	....	2	....	1	5
Miscellaneous	....	3	1	....	1	....	1	....	....	1	....	13	....	....	20
Undetermined	....	2	....	5	2	....	1	2	....	....	....	17	1	....	30
Total	23	46	15	13	21	4	25	20	2	16	5	126	6	10	332

REFLECTIONS.

Anyone who studies these reports, as the writer has, must be profoundly affected by them, particularly if he is at all sensitive to the unhappy experience which confronts the physician when one of his charges unexpectedly succumbs to an operation.

With this feeling I have set down what I term reflections, preferring to call them after this somewhat indecisive term rather than to hold them as well grounded conclusions.

They are presented in the hope that they may in some measure, if ever so slight, serve to reduce preventable fatalities after operations on the nose and throat.

I. AS TO THE PHYSICIAN.

1. Knowledge of the anatomy of the nose and throat cannot be learned by operating on the living patient—and yet, how many undertake to acquire it by surgery rather than by arduous study in the dissecting room.

2. To operate without this requisite knowledge is impossible for a man of conscience, not to mention the hazard to the patient.

II. AS TO THE PATIENT.

1. The patient himself, and not the disease or the name of the disease should be studied.

2. Is he a proper subject for the operation? Is he too young or too old? How are his arteries, his heart, his lungs, his blood and his kidneys? Has he tuberculosis, syphilis, diabetes, carcinoma or some eventually fatal chronic process? Is the operation likely to benefit him or is it worth while to operate upon him, considering his circumstances and environment, whatever such an operation may do for anyone else? These are potent questions of concern to the patient.

3. What might be termed the casuistry of operative indications is a good thing to study, for patients are not all alike; their response to operative intervention varies and the results, good and bad, cannot always be the same.

4. A good internist is a powerful help in the estimation

of the patient's reaction. He has saved and will save many a life for the surgeon.

5. Under all circumstances where it is possible, the patient should be hospitalized. We can count many indeed, from the reports, who could have been saved if the operation had been performed in a properly equipped hospital.

III. AS TO THE OPERATION.

1. Rigid attention to asepsis is essential, not by washing the field with antiseptic solutions or by applying iodin, but by operating with an aseptic organization such as would be used in any other important surgical operation. The nose tolerates its own bacterial flora without much reaction but not extrinsic bacteria.

2. The sitting position is bad for nose operations—the patient's head is too easily moved; fainting is common; toxic symptoms from cocain occur readily; the physician's arms are in an awkward position, and instrument control is not always secure.

The semirecumbent position is far better. The patient need not be held; the range of head movement is greatly restricted, fainting and cocain intoxication are uncommon, the surgeon has better control over the operative field.

3. It is better to take out too little than too much—even if a second operation be required. There would have been less fractures of the cribriform plate if all of the operators had followed this rule.

4. Multiple unrelated operations are an abomination—what is gained on the one hand is more than lost by the increased danger, the prolonged healing, the added complications. Furthermore, when untoward symptoms appear, who can tell which operation is responsible for them?

IV. AS TO THE POSTOPERATIVE CONTROL.

1. To operate and then to consign the patient to inexpert hands can have no justification. Many of the fatalities here recorded tell the tale of such practice, whether the surgeon leaves the patient, or the patient returns to his home without proper supervision.

2. The nurse or the mother or the attendant, whoever it may be, must be alert, particularly in caring for those who cannot explain their symptoms.

3. A better surgeon is required to supervise the after-treatment than to perform the operation.

V. AS TO THE FATALITIES.

1. Mankind has use for everything—even for adversity. Every fatality should call for an autopsy, carefully made and carefully recorded and then published, for the best way to avoid fatalities is to study those that occur.

2. The study and analysis of well kept histories and well planned autopsy protocols will bring about definite conclusions which will be of the utmost value in reducing the number of fatalities for which the less serious operations on the nose and throat are responsible.

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XX.

OPTIC NERVE AND ACCESSORY SINUSES.

By Prof. J. van der Hoeve,

Leiden, Holland.

Mankind is susceptible to inflammations of the nose. Every human being in his turn suffers from catarrh of the nose, some very often, others less frequently, but nobody is exempt from this trouble. Every time we have it, the inflammation may spread to the accessory sinus of the nose.

If we consider how very close and intimate the relation of these cavities is to the orbit and optic nerve, it seems almost a wonder that there are still some who have never had eye disease of nasal origin.

If out of the huge mass of eye diseases attributed to sinus affection we assemble those which concern the optic nerve, we must fix our attention upon several points.

1. The diagnosis of the optic nerve disease.
2. The diagnosis of the sinus affection.
3. The relations between these diseases.
4. The treatment.

THE DIAGNOSIS OF THE OPTIC NERVE DISEASES.

This diagnosis is less or more difficult according to the kind of nerve disease, which may be: choked disc, papillitis, atrophy and neuritis retrobulbaris Choked disc and papillitis, though it is not always easy to differentiate one from the other, are relatively easy to recognize, because the ophthalmoscope reveals in the very beginning that something is amiss with the optic nerve.

The diagnosis of the atrophy causes more difficulty, at least in the beginning, but the greatest difficulty is caused by the most frequent one, retrobulbar neuritis. The patients sometimes complain of pain behind the eyes in the depth of the orbit especially in movements of the eye. The ophthalmoscope is only of little benefit; in the beginning we see either nothing abnormal or a little hyperemia of the disc, dark and greatly

filled veins, until at last the characteristic pallor of the temporal quadrant of the disc appears. But then it is too late, the affection which could probably be cured before, is usually irreparable, therefore we must look for symptoms which enable us to recognize the affection in the very beginning when the treatment may be more successful.

It is possible that in the future the examination of the light perception will help us, but this has not yet been studied enough to be of much value and the best indices are given us by the examination of the visual field. Here we find sometimes as atypical symptoms concentric contraction of the visual field or annular scotoma and as characteristic signs: central scotoma and enlargement of the blind spot.

We do not at present know why the first loss of function appears in the peripapillar and central fibers; we have only to accept the fact.

We know that both scotoma begin as relative scotomata for colors only, whereas later the white light is also not observed. As the scotomata enlarge, they may unite and cause in this way the well known oval scotoma, including and surrounding the fixation point as well as the blind spot.

As a rule the enlargement of the blind spot appears first: if only one of the scotomata appears, it may enlarge until it invades the region of the other and causes the same oval scotoma, including blind spot and fixation point.

If we find this scotoma, we cannot say in which way it was formed but only this that the peripapillar fibers and the central fibers are affected.

Of course when we find one of these symptoms we must exclude the possibility of another origin, for the central scotoma may be caused by degeneration or hemorrhage in the macula, etc., the peripapillar scotoma by high myopia, medullar fibers, hysteria, etc.

Both symptoms are very important and we know with certainty that they can be caused by sinus affections, for I know of different cases of enlargement of the blind spot, as well as of central scotoma, where the scotoma decreased or vanished by treatment of the sinus affection and came back by every recurrence of the sinus diease.

We find the optic nerve affection much more frequent in diseases of the posterior than of the anterior sinuses, in which they are very rare.

Important as these symptoms are, we must be well aware that they are not symptoms of sinus affection but only of affection of the optic nerve.

They show that the optic nerve is diseased but they cannot teach us anything of the origin of this affection and we must always consider that every retrobulbar neuritis may begin with these symptoms, whether it is caused by multiple sclerosis, by tuberculosis, syphilis, dental infection, rheumatism or from a nasal affection.

So we arrive at our first conclusion:

The ophthalmologist has in the eye no sign to distinguish the origin of a retrobulbar neuritis.

DIAGNOSIS OF THE SINUS DISEASE.

Every rhinologist will be convinced that the diagnosis of an existing sinus disease, especially of a posterior sinus disease, is not always easy, we may say, not always possible.

So we find the following in Dan. MacKenzie's Diseases of the Throat, Nose and Ear, edited in 1920:

"The absence of any sign of disease inside the nose is not sufficient to exclude sinusitis from the diagnosis."

With the purely rhinologic methods of examination we are often not able to diagnosticate an existing sinus affection; happily we have support in the examination with Roentgen rays. We can photograph the sinus in different directions, occipitofrontal, when we wish to compare both frontal sinus, bitemporal and occipitocaudal, according to Pfeiffer's method, to compare the both sphenoidal sinus.

For the ethmoidal sinus I always think the method of Rhese to be the best.

The patient lies down on his tuber frontale, nose and ansa jugularis and the tube is put above the protuberantia occipitalis. Then we get in a normal person a photograph like this figure; you see the frontal sinus, the orbit and the region of the ethmoid; in this method we should always compare both sides.

For the ophthalmologist it is of great interest that we can get in this way an excellent image of the foramen opticum and

the fissura orbitalis superior, so that we can find out if something is amiss with the foramen opticum. In the various photographs the foramen appears as a nice round hole.

We photographed many persons with fractures of the skullbones and opticus atrophy in this way to determine if we could find a cause for the atrophy. In this are seen the callous on the base line of the skull and a foramen opticum which has an hiatus at the upperside and a fissure running from the foramen opticum in the skull. Here an underbroken base line and a callous on the foramen opticum. The base line can also be disturbed by other processes, for instance by a gumma, as in this case of Sonnenkalb. Here you see a foramen opticum which has quite another shape, after a skull traumatism. I use this method also to see whether the foramen opticum is free in patients with towerskull. It is said that the opticus affection in towerskull is caused by a deformity of the canalicus opticus, and Schloffer even operates on this theory to free the optic nerve in the canal. If I find a normal foramen I do not believe the canal can be much deformed, in fact, I have found a normal foramen opticum even in cases in which there was marked towerskull with choked discs. Care must be taken not to misinterpret a picture the result of wrong position.

Rhese's method demonstrates affections of the sinus frontalis as can be seen in this case of osteoma which overshadows the foramen opticum, but the great advantage of the method lies in the examination of the ethmoid, as in this case of obscuration of the ethmoid, in posterior ethmoiditis.

How important this method can be may be shown by the following case:

A physician had neuritis retrobulbaris and choroiditis; he was operated on for sinus disease several times by one of the best known professors of rhinology, until the professor told him, that though he did believe that he had not opened all affected ethmoidal cells he did not dare to go farther.

When I saw this man, I found a huge enlargement of the blind spot especially for colors and hyperemic disc. I informed him of this and advised him to have his nose treated again. The rhinologist, however, thought him to be a neurasthenic and did not wish to operate any more. The man went

to Dr. De Kleyn in Utrecht. Here Dr. Stenvers made a radiogram. The picture is shown here, and you see the whole ethmoidal region is clear except a rectangular spot close to the foramen opticum and fissura orbitalis superior. De Kleyn opened at that spot some posterior ethmoid cells in which he found purulent secretion and from that time the man was relieved of all complaint.

Thus we see the Roentgen photography may be of great support in the diagnosis of sinus disease and yet there are cases which are not discovered even with Roentgenography. The best proof of this is that the nine known cases of mucocele of the sphenoid sinus were not discovered before the sinus was opened.

In my case three rhinologists, among them the professor of rhinology in Leiden, declared that they could not find a single sign of sinus disease. The Roentgenogram was, as you see, not clear. Gerber supposed that this is caused by the extreme thinness of the bones surrounding the dilated sinus. When the professor of rhinology opened the posterior cavities on my advice, a yellow green mucous fluid streamed out, and then he could with probe reach the roof of the sphenoid sinus and discover that the roof was already perforated so that the sinus was in communication with the cerebral cavity.

MacKenzie also agrees that even Roentgen ray examination is not always sufficient, for he writes:

All cases must also be submitted to X-ray examination by a radiologist experienced in the skiagraphy of the nasal sinuses. But even X-ray examination is not infallible, so that in the presence of serious orbitoocular trouble, such as optic neuritis, for example, the only satisfactory method of excluding suppuration of the sphenoid and posterior ethmoid cells, the sinus most likely to set up eye disturbances, is to operate and open them up.

So we must say, as second conclusion:

The rhinologist cannot say with absolute certainty that a person has no sinus affection.

RELATIONS BETWEEN OPTIC NERVE AND SINUS DISEASE.

How is it possible that the optic nerve is affected by a sinus disease? What pathway is followed by the active agents?

To get an idea of this, we are in want of histologic examination of sinus, optic nerve and surroundings in such cases and it is a great pity we possess only very few of them.

As a rule the patients do not die from their sinus disease, and when a patient with a sinus disease and optic nerve disease dies it is very seldom that an autopsy is permitted where the part of the skull which we require is to be removed.

So there is little chance that the scant quantity of histologic material will be increased.

This chance will be better for material from sinus tumors than sinus inflammation, though the inflammation is more frequent.

Fortunately we have at least three histologic examinations which are all very important: two of tumors of the sphenoid, one of a pansinusitis.

It is highly interesting to compare the first two; in both cases there could be observed in vivo an axial neuritis which caused central scotoma for several months.

Birch-Hirschfeld found in his case the typical degeneration of the maculopapillar fibers, while on the contrary De Kleyn and Gerlach found in their case, though the central scotoma had already existed for more than seven months, almost no change in the optic nerve. They found the veins and capillaries of the optic nerve and nerve sheaths more filled than usual and a little infiltration of some of the nerve sheaths.

These two cases are typical of the clinical processes of the optic nerve disease in sinus affection, for we know we must distinguish here two forms, one which improves directly after the opening of the sinus, the other which does not diminish or diminishes only to a point and remains there.

In the first form there may be, as in the case of De Kleyn and Gerlach, only small and reparable changes in the optic nerve, in the latter irreparable changes, as in Birch-Hirschfeld's case.

The clinical symptoms in the reparable cases may be caused by edema, stowage, toxins, pressure and beginning inflammation, the irreparable by degeneration and atrophy as sequence of long continued inflammation or pressure.

De Kleyn and Gerlach's case is so highly important, because it shows that even after seven months of central scotoma nothing irreparable was found, so that we must never despair, be the duration never so long, but treat the cases as soon as we get a chance.

The other case of De Kleyn and Gerlach shows us how an inflammation can spread from the nose and nasal cavaties to the optic nerve and penetrate the bony separation wall. They found by histologic examination of a patient with pansinusitis ulcerations in the nose and lacrimal passage, infiltration around the epithelium of the mucosa of the sinus, and they saw, as I show you here, the infiltration penetrating into the narrow spaces of the bone and in other field the infiltration following a small vessel. Though by an unlucky accident a slide with sections was lost just at the field where the infiltration reached the optic nerve sheath, the other sections are so clear and distinct that we see how the inflammation takes its pathway from the sinus to optic nerve.

The optic nerve sheaths are infiltrated just at the spot where they are nearest to the sinus, and we see an interstitial neuritis optica start from the nerve sheaths.

The anatomic disposition may be of great influence whether an optic nerve will be diseased in the course of a sinus affection and we know, especially from Onodi, that the anatomic relations greatly vary.

Onodi showed us that the optic nerve may pass freely unprotected by any bony shield through an ethmoidal cell or even through the sinus sphenoidalis, while on the other hand in some cases the optic nerve is surrounded by a huge wall. This of course may be of great influence. If, for instance, a cell or sinus is filled with pathologic secretion the free optic nerve surrounded by this fluid may become diseased by it, and even if there is only slight or even no secretion at all, the nerve may still become diseased by direct contact, because it passes twice through the whole afflicted mucosa.

On the other hand, a swelling of the nerve may in sequence to toxic edema cause more disastrous effect if the nerve is enclosed in a bony canal; for the swelling may even give rise to such a pressure that choked disc or atrophy may result.

Moreover we know that the bony separation wall is not an absolute protection against invasion of inflammation, for we saw in the case of De Kleyn and Gerlach where pansinusitis gave rise to an optic nerve disease, that the inflammation can penetrate the wall and cause an inflammation of the optic nerve sheaths and of the nerve itself. So we cannot say that the protection of the nerve by a bony wall is an advantage in every respect.

As a rule we take it for granted, that the chance for the nerve is the greater the nearer it is to the diseased sinus, so that we even think that a sinus which touches the optic nerve of both sides or of the opposite side only, may give rise to affection of both nerves or of the opposite nerve. How many possibilities there are with reference to optic nerve and sinus sphenoidalis is shown by the schema of Quix and the preparations of Onodi.

That indeed the anatomic vicinity is of great importance is proved by De Kleyn and Gerlach's case where we saw the affection of the optic nerve begin at the spot where it is nearest to the sinus.

Thus we discover that there exist different ways in which a diseased sinus may affect the optic nerve.

1. By direct spreading of the inflammation, as is proved by the De Kleyn and Gerlach's case.

2. By pressure caused the walls of the dilated sinus, as we see in the cases of mucocele where every nerve in the neighborhood may become atrophic from the pressure.

3. We suppose that bad influence may be exercised by toxins, edema, stasis, etc.

We must distinguish:

a. Reparable optic nerve affections, which may remain for months and months, caused by toxins, edema, stasis, slight inflammation, slight pressure, and

b. Irreparable optic nerve changes caused by degeneration and atrophy, in consequence of the same processes as mentioned above, but greater in degree.

To get a better view of the way in which the sinus disease involves the optic nerve we stand in great need, not only of more histologic material of sinus and optic nerve disease after the atrophy, but also of bacteriologic and histo-

logic examinations of the material which the rhinologist gains by the operation upon the sinus, and it is to be hoped that every rhinologist will try to help close this unnecessary gap in our knowledge.

TREATMENT.

Here we must distinguish those cases of optic nerve affection in which there is a sinus disease affection and those which are of unknown origin, and where no sinus disease can be diagnosticated. The first cases are easy enough. Where we find a sinus affection in a patient with optic nerve disease we must treat the sinus. We know that a sinus disease may cause an optic nerve affection and therefore we have the right to suppose that if a sinus disease is present in a patient with an optic nerve affection of another origin, the latter may be aggravated by the former. Where this is the case we ought to treat every sinus affection in a patient with an optic nerve disease, even if this is not of nasal origin. I always should advise ophthalmologists and rhinologists to begin their treatment in a conservative way; many sinus diseases are cured with the cocain adrenalin spray.

The other category causes us on the other hand very much difficulty.

What to do when a patient has an optic nerve disease and the rhinologist does not find by careful examination the signs of a sinus disease, Roentgen rays included.

Of course the patient must be examined carefully for internal and neurologic diseases. When some other cause for optic nerve disease is found, I should still always urge, to consider the accessory sinuses. It is possible that a patient with multiple sclerosis, with diabetes, syphilis, tuberculosis, rheumatism or post trauma may also have a sinus disease. It would be very disagreeable to be contented with one cause for the optic nerve affection and overlook sinus disease which might be the real cause or an adjuvant. I remember a patient who was sent to me by an ophthalmologist, for optic nerve disease and purulent secretion in the nose, but the rhinologist refused to open the posterior sinuses because he did not find other signs of sinusitis and the patient had syphilis. According to the rhinologist the origin of the nerve disease was absolutely explained

by the syphilis. They used antiluetic treatment alone until one eye of the unlucky patient was absolutely out, and the other almost blind. In Leiden, the rhinologist could find no other affections of nose and accessory sinus in the nose but when he opened the posterior sinuses of both sides, on my advice, there was purulent inflammation of both sinus sphenoidales and the posterior ethmoid. Soon after this the visual acuity of one eye recovered, becoming more than 1/2, but several scotomata remained and the other eye remained blind. A patient with multiple sclerosis, syphilis, tuberculosis, rheumatism or trauma may suffer from a sinus disease which has nothing to do with the affection, but he may have a sinus disease caused by the syphilis, the tuberculosis, the rheumatism or the traumatism; so that these diseases are not the direct cause of the optic nerve disease but the indirect, whereas the sinus affection is the direct cause. Therefore we ought always to examine the nose and accessory sinus in every patient with optic nerve affection, even if there is found another possible cause for this affection. When nothing at all is found, many authors say it will be multiple sclerosis because this disease is thought to be in 50 to 70 per cent the real cause of retrobulbar neuritis and the optic nerve affection may precede every other symptom 10 or more years. Even if this is right we cannot let our treatment be influenced by this consideration.

When there is an optic nerve disease without any other symptom of multiple sclerosis, for us the patient has no multiple sclerosis, at least we cannot perceive it, and we must treat him as if he had no multiple sclerosis until he gets other symptoms of this disease.

What to do in those cases of optic nerve disease without known origin is very difficult to determine, and a very close cooperation of rhinologist and ophthalmologist is required. We see in other countries how necessary people think it to discuss this problem in joint assemblies of rhinologists and ophthalmologists, as was done in September of last year at Nauheim in Germany; in March, this year, in Vienna, Austria, and in May, in Paris, France. You Americans have the good luck to possess this academy, so that you can discuss this and other problems together as often as you like to do.

Now, there are ophthalmologists who say that when the ophthalmologist thinks it necessary to open the sinuses the rhinologist must obey; he is in this way the handcraftsman for the ophthalmologist.

I am not of that opinion. I for one think that an organ specialist is not only the man who knows most of his special organ, has the most practice in operating on it, but is also the man who must watch his organ as a precious treasure which is under his trust, so that he must guard it from unnecessary mutilation.

No surgeon of whatever kind has the right to operate, that means to mutilate an organ, when he is not thoroughly convinced that what he is doing gives the best chance to the patient; he must never operate on the responsibility of another doctor. This is as well the case in appendicitis, ulcus ventriculi, trepanation for tumor cerebri or in sinus disease. Of course the surgeon cannot master every special method of examination and so he must believe the internist when he tells the surgeon what he finds upon examination of the stomach and intestinal contents, the ophthalmologist when he tells him of the visual acuity, the visual field, the choked disc, the neuritis, etc., but when he examines the patient himself, and when both have communicated with each other what they found by their examination and what they think of the diagnosis then they must consult with each other about the possibility and the desirability of the operation in general and in every individual case. When the surgeon is convinced by the result of his own examination or by the arguments of the other physician, he has the right to operate, otherwise never.

We now return from these general remarks to the optic nerve and sinus diseases. We know that our treatment will be greatly influenced by the conclusions already made.

1. The ophthalmologist cannot say whether or not an optic nerve disease is of nasal origin.

2. The rhinologist cannot exclude with certainty the presence of a sinus disease: to those two we add:

3. Opening of the sinus, even if nothing pathologic is found in the sinus, may be of temporary or permanent good effect on the optic nerve disease.

I think this latter result may be produced by derivative treatment; it may be that the bleeding or the change of the lymph stream is the cause, so that it acts as a bleeding or as installation of dionin or subconjunctival injections.

When we ask the ophthalmologist must the sinus be opened and he views the question only from his narrow point of view as organ specialist, he must say yes, as the eye has nothing to fear from such an operation and much to gain, whether or not a sinus disease is discovered.

If, on the other hand, the rhinologist is asked, must we operate? He must say as organ specialist no, as the nose is mutilated by the operation and there is only a little chance that the nose has to gain anything, that is, in the case that a sinus affection is discovered. Then they must consult together, but the ophthalmologist should not say, as v. Eicken describes, this patient will soon get blind, if you do not open the sinus and his eyes will be saved if you do. Then the rhinologist has no choice, but he is persuaded by unfair means, as no ophthalmologist can say whether a patient will get blind or will stay blind, or if he will recover after the operation.

Our treatment will of course be influenced by the eye symptoms; if a patient is blind or nearly blind no rhinologist will refuse to give him a last chance, but the greatest difficulty is in the chronic cases.

Here the rhinologist must ask the ophthalmologist what can be gained.

What do you know from literature as to the frequency and the amount of recovery of the visual power after the operation and of the number of cases in which a sinus disease is found and what is your personal experience in this? They know what is gained usually and what is the personal experience of the ophthalmologist. On the other hand the ophthalmologist must ask the rhinologist what is the damage which can be done by the operation. I know that it is possible in an imprudent operation to curette away an optic nerve which passes freely through a sinus; this can be prevented. In what per cent is damage done after the sinus operation, and what kind of damage, and what is your personal experience? The per cent figures gathered from the whole literature may decide whether in general the operation

is tolerated. Say for instance that in every 10 sinus operations in optic nerve disease of unknown origin we find once a sinus disease and once the visual power is considerably increased, then we must ask ourselves whether we have the right to operate unnecessarily on eight patients to help two. The personal experience of both specialists is of influence too, as the experience of the ophthalmologist shows whether he has a good clinical view in choosing the patients upon whom an operation is to be performed, the experience of the rhinologist showing whether he has good luck as operator or not.

If they both know the possible advantage and possible damage, they have to decide whether or not in a special case they will operate and in this regard the personality of the patient will be of value, as we must always keep in mind that a sinus operation is not an indifferent matter for the patient.

Though we can make the nose tolerably anesthetic, every patient thinks a sinus operation is very disagreeable and for a nervous patient it is a terrible shock, so that we cannot deny the influence on the psyche, especially when the operation has no effect. In strong minded people we may discuss as far as possible the question with the patient himself, in others with the family. Thus one of them should know that there is no question of an operation for an existing disease but an exploratory operation for a possible disease, otherwise they will always think if no good result is gained that the operator failed to do his duty.

Acting in this way I have never met a rhinologist who refused a sinus operation when I wished him to do it and I may say that I was very lucky in the results, so lucky that I hope I will always have the same experience.

You see from these considerations what is failing; we want urgently, statistics of the results of the operation as well as to the direct and late results for the visual power and of the results of the damage to the nose, but also statistics of what happens with the eyes of the patients in which the sinus is not opened. To make these statistics it is necessary that we all use the same terms, which I am not convinced has applied. We see that in Nauheim Von Eicken compares statistics of Heine in Kiel, who found in 50,000 patients 46

cases of retrobulbar neuritis, that means in 0.092 per cent, and among them only 3 cases of nasal origin, that means 0.006 per cent, and the statistics of V. Grosz in Budapest, who found in 18,587 patients 58 cases of optic nerve disease of nasal origin, that means in 0.312 per cent or 52 times as frequent. V. Eicken suggests that this considerable difference may be caused by the fact that V. Grosz perhaps reckons to be of nasal origin every case in which the visual power is increased by an endonasal operation. But if this was the case it must still be explained how it is possible that V. Grosz has 3.5 times more optic nerve disease of nasal origin than Heine in retrobulbar neuritis of all origins. Either the optic nerve disease must be much more frequent in Budapest than in Kiel, or V. Grosz and Heine do not use the same terms.

So I think the first thing necessary is to get uniformity in the nomenclature and then try to get statistics of the advantages which the eye gets from the sinus operation and of the damage which the nose gets from this operation.

I hope, Mr. Chairman, ladies and gentlemen, that you will all help to develop such statistics in order to solve the highly important question, as to whether we must operate upon the sinuses in optic nerve disease of unknown origin.

XXI.

RECENT OBSERVATIONS ON LARYNGOPULMONARY TUBERCULOSIS.*

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We all realize how difficult it is to render an accurate prognosis in laryngopulmonary tuberculosis. The average length of life of a patient suffering from laryngopulmonary tuberculosis has been estimated by various men to be from two to four years. Although this may be a fact, to the individual patient it means very little, for we all know that some cases of laryngopulmonary tuberculosis live but a few weeks, while others enjoy good health for many years. The individual affected is not particularly interested in the average length of life, but he does want to know how long he may expect to live. In order to arrive at a fairly accurate prognosis it behooves us to study each case by itself, and even then we may expect to err occasionally.

It is generally understood that laryngeal involvement renders the prognosis very serious. Older writers have been of the opinion that laryngeal involvement rendered the prognosis absolutely hopeless. My personal observations are at great variance from the generally accepted opinion, and it is my present purpose to bring forth some of my observations based on nearly ten years' experience with tuberculosis of the lungs and of the larynx.

While on the staff of the New York City Sanatorium at Otisville, New York, from 1913 to 1918, I observed a large number of patients with laryngeal tuberculosis, some of whom lived only a short time, while others lived and enjoyed good health for years in spite of the fact that their lesions were quite extensive. This puzzled me very much, and therefore I examined a large group of patients, carefully noticing the

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pathologic manifestations. I also observed these patients for a long period, making careful record of the course of the disease in the larynx. From these observations I became convinced that laryngeal tuberculosis was either peracute, acute, subacute, or chronic. These various forms have already been discussed by me in previous works, but because of the intimate bearing on the subject I will describe them again.

The peracute cases of laryngeal tuberculosis, fortunately, are infrequent compared with any of the other forms. These cases are very rarely seen in the stage of edema, but usually present extensive ulceration and perichondritis. They are often accompanied by tuberculosis of the faucial tonsils and soft palate. Dysphagia and aphonia are always present, especially when the pharynx is involved. Emaciation is rapid, the patient is febrile and soon becomes moribund. Other organs of the body, such as the intestines and meninges, are often also involved. The peracute type usually begins as such, although occasionally it follows the acute type.

The acute type is characterized by a soft edema of the larynx with a tendency to ulceration. It may begin as such, or it may follow the subacute or chronic type. It usually occurs in the advanced cases of pulmonary tuberculosis, often in moderately advanced cases, and occasionally in incipient cases. There is little or no tendency to fibrosis. Nodules consisting of tubercules can often be seen projecting above the surface of the mucosa. When the tubercles caseate they give rise to ulcers. During the stage of caseation the nodules often coalesce, with the production of extensive ulceration. The patient usually suffers from marked hoarseness or aphonia, a sensation of fullness in the throat, and irritation with a constant desire to expectorate. Dysphagia is of common occurrence and may be very pronounced, depending on the location and character of the ulcerations.

The subacute type is characterized by a semiedema of parts of the larynx with a moderate tendency to fibrosis. It may begin as such or else follow the acute or chronic type. It takes a subacute course, has a tendency to healing and is usually benefited by treatment. Lesions with papilliform infiltrates and soft polypoid excrescences generally belong to this type. The local symptoms may be slight or moderate, de-

pending largely on the site and the extent of the lesion. Hoarseness, as a rule, is present, particularly when the true vocal cords or the interarytenoid sulcus is involved. Dryness of the larynx is generally complained of, and more so when the fibrosis is extensive.

The chronic type offers the best outlook, and is characterized by firm infiltration of part or parts, with a marked tendency to fibrosis and healing. The lesion, due to an extensive proliferation of connective tissue cells, is usually limited in extent to the affected areas, and may remain so for an indefinite period. These patients suffer very little, and often, with the exception of occasional dryness in the larynx and slight hoarseness, have no symptoms at all. For this reason this type is very often overlooked.

In rendering a prognosis in laryngopulmonary tuberculosis, although all other factors, such as character and extent of the pulmonary lesion, the general condition and mental condition, early diagnosis, etc., should be considered, the chief determining factor is the character of the laryngeal lesion itself. For it very often happens that a patient will be given a good prognosis based on his pulmonary lesion, only to have the prognosis changed when it is discovered that he suffers from an acute involvement of the larynx as well.

The summary of my observations on the prognosis in laryngopulmonary tuberculosis, therefore, is as follows:

In peracute cases, with or without involvement of the pharynx and regardless of the pulmonary lesion, the prognosis is bad, and the life of the patient is from a few weeks to a few months. Here, as a rule, the patient dies from an overwhelming toxemia and from inanition caused by dysphagia. Local treatment is of no avail and is not indicated except for the relief of pain. Our aim should be to make the patient as comfortable as possible.

Acute cases often improve under skillful treatment, and when the pharynx does not show macroscopic involvement and the pulmonary and general conditions are at all favorable, the prognosis is not hopeless. These patients should be kept under a careful regimen and persistently treated generally and locally. They often improve and occasionally become completely cured.

The subacute cases present a good outlook, and the prognosis depends a great deal on the pulmonary lesion and timely treatment. With good general care and with proper local treatment the laryngeal lesion becomes fibrosed and chronic.

The chronic cases present the best outlook. Patients with this type of lesion usually get along best under general treatment alone. The prognosis as to life is hardly ever influenced by chronic laryngeal tuberculosis, and therefore in rendering a prognosis in these cases the larynx may be entirely disregarded.

I shall now illustrate my paper by demonstrating to you a few plates of the different types of laryngeal tuberculosis:

Case Record No. 1.—Mrs. R. G., age 20. Patient consulted me on account of slight hoarseness and dysphagia. Examination revealed early pulmonary tuberculosis. somewhat active. Larynx showed edema of epiglottis and both vocal cords, and a semiedema of both arytenoids. Patient also gave a history of a six months' pregnancy. General and local treatment were instituted but apparently without result. Dysphagia became worse and emaciation progressed. About two weeks later both tonsils revealed the presence of macroscopic tubercles and gradually the soft palate and uvula became involved. The disease progressed rapidly and patient died about twelve weeks from the time I first saw her, giving premature birth two days before death.

Diagnosis: Peracute tuberculous laryngitis with involvement of tonsils and soft palate. Fig. 1.

Case Record No. 2.—R. M., aged 36. Pulmonary lesion advanced and active with bilateral involvement. Larynx showed very acute edema of epiglottis, arytenoids and aryepiglottidean folds. Patient has a great deal of dysphagia and suffers from complete aphonia. Local treatment has been attempted but with no results. Diagnosis: Peracute tuberculous laryngitis; prognosis, very bad. Fig. 2.

Case Record No. 3.—E. N., age 23. Patient was referred to me by Doctor Thomas R. French, of Brooklyn, New York, in February, 1921. First examination revealed that he suffered from advanced pulmonary tuberculosis with activity. He ran an afternoon fever of 101-102; was considerably emaciated and suffered from loss of appetite. Examination of

larynx showed acute edema of epiglottis with angry looking ulceration of right side. Rest of larynx seemed quite free. Dysphagia was pronounced. Patient was put to bed where he was kept for about four months and larynx was treated locally. Examination in early part of September showed that pulmonary lesion was quiescent; temperature was normal; patient gained about five kilograms in weight; ulcer of epiglottis was almost completely healed, with some loss of tissue and formation of scar; dysphagia completely relieved and patient allowed to return to work. Diagnosis: Acute tuberculosis of larynx, healing by fibrosis. Fig. 3.

Case Record No. 4.—M. H., age 30. Referred to me by Doctor V. G. Bourke, of Livingston Manor, New York. First examined February 2d, 1921, and her condition was as follows: Pulmonary lesion—Far advanced and moderately active; sputum positive. Larynx revealed inflammation and uniform infiltration of epiglottis with ulceration; infiltration with erosion of both aryepiglottidean folds; edema in the posterior commissure and pear shaped swelling with ulceration of both arytenoids. There was also an acute infiltration of posterior ends of both vocal cords. Patient ran a fever in the afternoon and suffered from great dysphagia and aphonia. The laryngeal lesion was apparently of the acute type. General treatment was instituted, and in addition to vocal rest the patient was treated with topical applications of solution iodin. On October 5, 1921, patient showed general improvement; pulmonary lesion was considerably more quiescent; examination of the larynx showed the epiglottis only very slightly edematous, and on a casual examination it might even be considered normal; the arytenoids showed only a slight thickening, with healing ulceration. All the edema observed eight months ago was practically gone. No dysphagia, and patient now has a pleasant low pitched voice. Diagnosis: Acute tuberculosis of the larynx, gradually healing by fibrosis. Fig. 4.

Case Record No. 5.—J. F., age 24, is a case of far advanced pulmonary tuberculosis with positive sputum; marked hoarseness. Examination revealed extensive infiltration of both cords with large papillomatous mass in posterior commissure, interfering with complete approximation of cords. Diagnosis was subacute tuberculosis of the larynx. Patient has been

treated locally with topical applications of solution iodin in increasing strengths and is showing steady improvement in voice. Fig. 5.

Case Record No. 6.—L. R. Patient has a moderately involved pulmonary lesion with positive sputum. Examination of larynx shows a papillomatous growth in posterior commissure which has been stationary for over a year. He is practically free from symptoms, except for occasional discomfort and feeling of irritation in larynx. Such a lesion practically never influences the prognosis. Diagnosis: Chronic tuberculous laryngitis, with a good prognosis. Fig. 6.

Case Record No. 7.—M. F., aged 19. Referred to me by Doctor Thomas R. French, of Brooklyn, N. Y. First examined on March 12th, 1921. Findings were as follows: Pulmonary lesion incipient and inactive; sputum positive; examination of larynx showed a warty growth of epiglottis and a general hyperplasia of the mucous membrane of the entire larynx, with thickening of the arytenoid cartilages; a small portion of the rim of the epiglottis was recently excised for diagnosis, leaving a large slowly granulating ulcer. Patient suffered from much dysphagia. The patient, in addition to general treatment, was also treated by local applications of solution iodin, and on September 2d the examination showed the epiglottis much diminished in size and edema generally lessened. The entire larynx showed distinct improvement with complete relief from dysphagia. Diagnosis: Lupus of larynx, gradually healing. Fig. 7.

Case Record No. 8.—J. C., age 38. Admitted to the Otisville Sanatorium in 1913 and was found to have a moderately advanced pulmonary lesion with a positive sputum. Wasserman negative. Larynx showed a tuberculoma of interarytenoid space with healed erosion, thickening and infiltration of both cords, especially the left. Left cord also showed healed ulceration on its upper surface, infiltration of both ventricular bands, especially the right, which almost entirely covered the right cord. Lesion showed complete fibrosis, and outside of marked hoarseness patient had no discomfort. The diagnosis was chronic tuberculosis of the larynx.

Examination of the larynx three and one-half years later showed the findings to be identically the same. In the mean-

time the patient has been doing very well and shows no signs or symptoms of decline. The probable duration of the lesion according to the patient's history is about ten years. The patient does not receive any local treatment.

I have not seen this patient in the past four years, but in answer to an inquiry from me, received last week, he states that he works, has gained some weight, and feels as well as ever. His voice is still husky, but with the exception of occasional irritation in the throat he does not experience any troublesome symptoms. Fig. 8.

Case Record No. 9.—I. T., age 20. Admitted to the Otisville Sanatorium on December 26, 1916. Chest examination revealed a moderately advanced lesion of the lungs. On the third day at the sanatorium, the patient developed a slight afternoon fever, which afterwards became persistent, rising to 101-102° F. on every afternoon, while the pulse ranged between 110 and 130 during his entire stay at the sanatorium. The examination of the larynx, on admission, showed a uniform infiltration of the epiglottis, with slight thickening at the posterior commissure and arytenoids. On January 5th the patient complained of slight dysphagia, and the examination showed the beginning of tuberculous infiltration of the oropharynx which kept getting more extensive, while the symptoms were gradually becoming more troublesome. On February 1st the patient complained of severe dysphagia and regurgitation of liquid food through the nostrils. He then showed extensive infiltration and ulceration of the oropharynx and fauces. Uvula was edematous. Larynx showed edema of epiglottis and both arytenoids, and the Wasserman test was negative.

On February 14, 1917, the lesion was widespread. Uvula was much swollen and covered with macroscopic tubercles. The lingual tonsils were also ulcerated. The larynx showed acute edema with macroscopic tubercles of all parts. All during his stay at the sanatorium the patient received local applications of solution of iodin to the affected parts, with no apparent benefit. On February 15th the patient was transferred to a city hospital, where he died a few days later.

This is a typical case of the peracute type and the prognosis is extremely grave. Fig. 9.

Case Record No. 10.—C. B., age 20. Pulmonary lesion moderately advanced with positive sputum. Laryngoscopic examination showed infiltration with superficial ulceration of the epiglottis; also infiltration with ulceration of both true cords ventricular bands and arytenoids. There was also marked interarytenoid hyperplasia with ulceration. Patient suffered from extreme hoarseness and sense of fullness in the larynx but there was no dysphagia.

The second picture shows the same patient after seven months of treatment. The edema had almost entirely disappeared; ulcerations, with the exception of those in the posterior commissure, are all healed. There was marked improvement in phonation, and the lesion showed steady healing by fibrosis.

The same patient was seen by me recently, about seven years after my first examination. The appearance of larynx is about the same as in the second picture; the epiglottis is absolutely free from involvement; the patient is still somewhat hoarse, but generally seems to be in fairly good condition Diagnosis: Acute tuberculosis of the larynx which is gradually healing by fibrosis. Fig. 10.

Fig. 1.

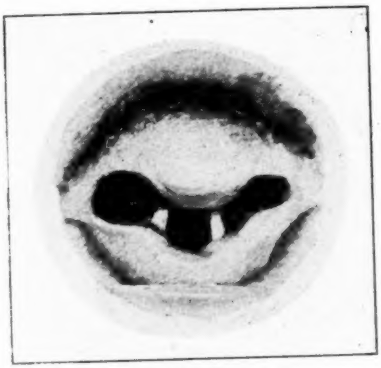

Fig. 2.

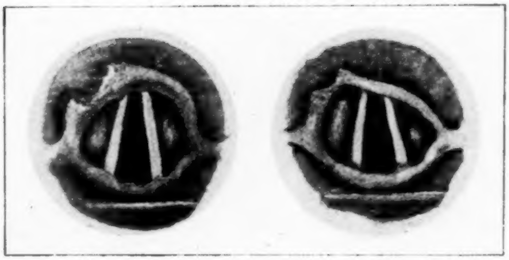

Fig. 3.

Fig. 4.

Fig. 5.

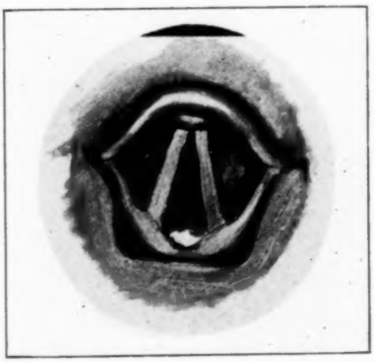

Fig. 6.

Fig. 7.

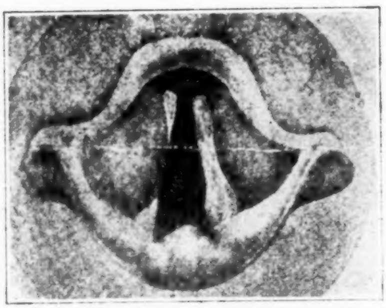

Fig. 8.

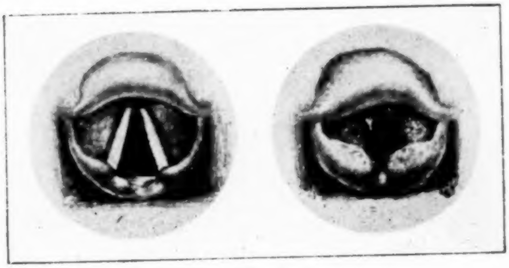

Fig. 9.

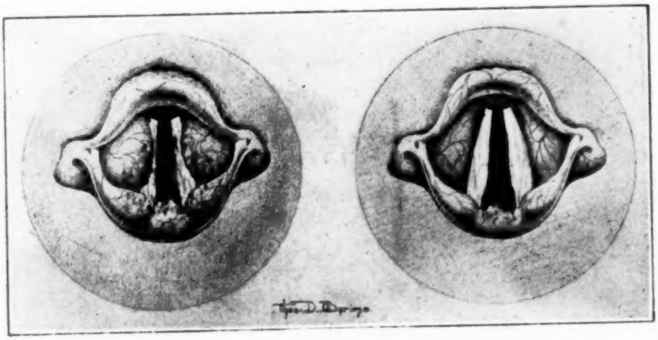

Fig. 10.

XXII.

THE USE OF EAR PLUGS IN AVIATION.

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Mitchel Field, L. I., N. Y.

Everyone who has flown for a considerable period of time, especially in bimotored ships, is familiar with the bothersome after-ringing in the ears and partial deafness lasting for several hours. These disturbances are caused by the sharp crack of the exhaust from the airplane motor, and if this condition is endured there will develop, eventually, more or less permanent impairment of hearing, inasmuch as the inner ear is a delicate organ and will not tolerate abuse without showing effects. The temporary effects of this exposure of the ear to massive sound are the ringing and temporary deafness, as stated above. In addition, however, if the abuse is continued, the temporary deafness may result in permanent partial deafness. It would seem wise, therefore, to remedy this condition, if possible, in order that the air life of the flier may not be shortened.

Three possible means of accomplishing this present themselves. One is the abolishment of the exhaust noise of the motor by a muffler, inasmuch as this sharp crack of the exhaust is the sound that causes the trouble. The remaining noise of the motor, the noise of the propeller, struts, etc., is either too low pitched or of not sufficient intensity to be harmful. Airplane motors have been successfully muffled, but at the expense of some of their efficiency, so that fighting planes will probably remain unmuffled. Secondly, a cabinet, nearly soundproof, can be constructed which will eliminate enough of the noise. This has also been done, but again at the expense of the efficiency of the airplane. It would seem, there-

fore, that at the present time nothing can be done with the airplane itself to eliminate enough of the motor noise. This being true, the fliers themselves offer the only means of abolishing this menace. For this reason, various substances were placed in pilots' ears to determine if one could be found which would not interfere with the flier hearing his motor, which would not hurt or annoy the pilot in any way, which could be easily and readily inserted or removed, which was reasonably inexpensive, and which would eliminate enough of the sound so that the flier would have no bothersome after-ringing or deafness.

Most fliers use cotton in their ears. This helps some, but does not usually abolish sufficient sound, and becomes easily displaced. Rubber plugs of various sizes and shapes satisfy certain fliers, but, due to the difference in the shape of the ear canal, a plug that will fit one man's ear will not necessarily fit the ear of another, hence, if used, will result in pain, discomfort or displacement if too large, and in insufficient protection and displacement if too small. In fact, any solid substance which cannot be moulded to the outer ear canal, even if composed of material as soft as rubber, must, in order to be efficient, be placed in so tightly that it will cause irritation of the delicate membrane lining the canal. Consequently, research work was undertaken here to find some substance, pliable enough when warm, to mould itself into the form of the outer ear canal and hard enough to retain its shape after being so moulded. Various substances, like parresine, beeswax, printer's wax, and gum were tried. The Navy Aviation Medical Service had previously tried parresine alone with some success. We found here that better results were obtained with a mixture of parresine and beeswax: five parts of parresine with one part of beeswax for summer use, and four parts of parresine with one part of beeswax for winter use, as beeswax softens the mixture and more of it is necessary in colder weather. Ear plugs of this mixture seemed of the right consistency, but lost their form too readily; hence, solid substances were used as a core to give "body" to the plugs, make them hold their shape better, and permit of easy withdrawal. Absorbent cotton does not possess the necessary resiliency and becomes too easily packed. After considerable experi-

mentation it was discovered that lamb's wool (surgical wool) was ideal. It does not pack as easily as cotton, yet is readily moulded; consequently the plugs hold their shape better and can be easily withdrawn.

In order that a large number of plugs can be made at one time, moulds were prepared from plaster of Paris blocks. These moulds are in two sections, thus enabling the completed plugs to be easily removed. Figure 1 shows a photograph of one of these sections, and from it some idea of the shape of the plugs can be ascertained, as well as from Figure 2, a photograph of an actual plug.

In making the plugs we first grease the moulds with a thin coating of vaseline; this prevents the wax from adhering to the mould. The two sections were then apposed and a small tuft of lamb's wool is placed in each depression. The melted mixture of parresine and beeswax, in the proportions required, is next poured in and allowed to cool, after which the moulds are separated, the plugs removed and any irregularities in their shape smoothed off. For shipping, the plugs are packed in an ordinary pasteboard box.

The plugs must be warmed and moulded before inserting. This is accomplished by holding a plug in the palm of one hand and kneading it with the fingers of the other hand. When it is soft enough, it is moulded into about the form depicted in Figure 3, and is then ready for insertion. When the plug is placed in the ear, the main thing to be observed is to avoid having the conical end so long that it will impinge upon the ear drum. This will not do any material damage but will cause discomfort when inserted. The plug is firmly inserted with the conical end in the external ear canal, and the remaining flat portion moulded over the end of the canal in the ear outside. A pledget of absorbent cotton, or, preferably, wool, about the size of a small egg is next placed in the ear on top of the plug and the helmet drawn down over this. This cotton or wool serves the purpose of maintaining constant slight pressure upon the plug, thus preventing its becoming dislodged by vibration. Withdrawal of the plug is easily accomplished by engaging any portion of it with the finger nail. Figure 4, the photograph of a withdrawn plug, shows the general shape which the plugs assume in the ear.

Several hundred of these plugs were sent to each of the flying fields throughout the country, and the Flight Surgeons of these fields were requested to report upon their use, adding any suggestions or criticisms that they or others cared to offer which would make for a better plug. A number of letters have been received from these Flight Surgeons stating that while the plugs are not in universal use at their fields, those fliers who had used them were very much pleased with them. A number of pilots still share the belief that any artificial means tending to preserve their health must not be used because they might be adjudged to be "soft" or timorous. This attitude, however, is becoming less and less prevalent, and it is believed that in time ear plugs of a type similar to those described above will constitute part of the equipment of every flier.

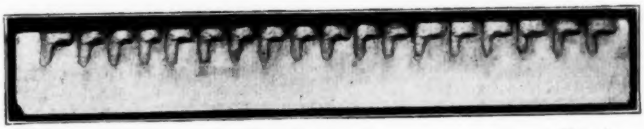

Fig 2. Fig. 3. Fig. 4

Fig. 1.

XXIII.

LARVAE OF THE FLY SARCOPHAGA (SARCOPHILA) WOHLFAHRTII IN THE EAR.

By Alexander Zebrowski, M. D.,

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It is well known that the number of foreign bodies, both animate and inanimate, met within the external ear channel is very great. In every textbook of otology we find enumeration of all possible foreign bodies found within the external ear. Kerrison[1] in his book says "there is hardly a limit to the variety of foreign substances which may be introduced, either intentionally or by accident, into the external auditory canal." In the chapter on the foreign bodies in the ear the authors all emphasize the importance of skill and care in the removal of foreign bodies, stating that no foreign body can do so much harm as a careless and unskilled trial to get the foreign body at any price. Except in the textbook of Ballenger[2] I have nowhere found a notice that an animate foreign body—e. g., the larvæ of insects—can occasionally endanger the life of its victim, although in the majority of textbooks on otology we find mention of larvæ in the human ear.

As a curiosity I cite a statement of such an authority as Bezold,[3] who in his textbook denies the possibility of the presence of larvæ in the human ear.

Nevertheless, it is a fact that at least one kind of fly, namely Sarcophila or Sarcophaga, described at first in the eighteenth century by a German physician, Wohlfahrt,[4] can cause most severe pain and under certain circumstances may endanger life itself. Wohlfahrt had a patient 67 years of age, who for eight days suffered horrible pains in the head. The regions of the right eye and nose were swollen. There was epistaxis from both nares which lasted several days without cessation. One larva fell from one nostril, and when the patient, according to the advice of Wohlfahrt, poured some alcohol into the nose, 18 larvæ came out and all symptoms disappeared very soon.

Wohlfahrt examined the larvæ, and after some weeks of waiting there appeared a cocoon and a fly, which he has described and very instructively illustrated (Fig. 1). This observation for a long time remained unknown and the German entomologist Schiner described the same fly in 1869 under the name Sarcophila magnifica, without mentioning the description of Wohlfahrt. A prominent Russian entomologist, Portchinsky,[5] claimed that this fly should bear the name of the discoverer and proposes to give her the name "Sarcophila Wohlfahrtii," or "Wohlfahrtia magnifica." In his very interesting monograph he gives all the data from the literature, describes the fly, its customs, and cites a great number of cases where the larvæ have penetrated into the human body, for the most part, however, into the ears, nose and eyes.

The fly is very common in Southeast Russia, in Southern Siberia and Turkestan. Its habitat is near herds of cattle, chiefly sheep, on which it deposits its larvæ (not eggs, but larvæ), for the most part in even the smallest wounds. The cattle owners in that part of Russia know very well the dangerous "worms" on cattle, from which the animals sometimes die, but, according to Portchinsky, they did not know that the worms are descendants of a beautiful big fly, grayish in color, which Portchinsky has repeatedly found in the neighborhood of the cattle. The fly appears only on hot sunny days; at other times it is hidden The female flies have a very delicate sense of smell, and even the smallest wound attracts them. No wonder that when a child with a purulent discharge from the ear is sleeping in the open the fly, when present, will come very quickly and put her larvæ into the ear.

The larvæ when born are small, but very active, and grow rapidly. They have on the head end an apparatus consisting of three strong sharp hooks (Fig. 2), with which they penetrate into the tissues of the body and make deep, long channels, causing inflammation with profuse discharge. This discharge serves as food for the larvæ. In the second stage of the development they lose the middle hook, become quite large (up to 15 mm. length) and then leave the animal. Then they burrow in the ground, form a "false" cocoon and in two weeks a fly is born.

After this introduction I shall turn to the description of the cases observed and treated by me in the years 1915-16, when I was practicing in the Crimea. My first case came to me in September, 1916 (this month in the Crimea is very hot). During my office hours a whole Tartar family came to me, bringing a boy 10 years of age who was crying, yelling, groaning and clutching at his head. Sometimes he was even swooning. The parents told me that for four days the boy had not slept a wink, complaining of terrible pains in the ear, which had been discharging pus for three years (after measles). They had seen what they described as large worms in the ear. They removed some of them, but some are still there, and the country physician could not get them out by syringing with water.

The aspect of the left ear was awful. The whole auricle was reddened, swollen and very painful to the touch. In the meatus several larvæ were to be seen. When I tried to pick some of them with the ear forceps they burrowed quickly into the depth of the meatus and other larvæ came into view. The tragus and lobulus were covered with pus, blood and black spots. The mastoid process was tender on pressure, especially in the fossa mastoidea. The lymphatic glands on the neck was enlarged and swollen. Temperature, 102°. The pulse, weak, 130. The boy appeared to be very ill.

At first I tried to syringe the ear with water, but only one larva came out. Seeing such a thing for the first time in my life, I examined it with the magnifying glass and discovered the hooks mentioned above. The larva made quick movements with the head and whole body which did not cease even when I put the larva into H_2O_2 and alcohol. Knowing that olive oil makes the removal from the ear of living insects (bugs, cockroaches) easier, I filled the ear with oil, which I always have on my office table for softening hard cerumenous plugs. Some seconds after that the larvæ appeared, one after the other. The oil had closed their breathing organs, forcing them to escape from the ear channel. Altogether, eleven larvæ, about 10 to 12 mm. long, came out. The child at once became quiet and fell asleep. After careful cleansing I examined the ear and found very remarkable changes. The skin layer of the auricle and external ear channel was red and swollen, with small

excoriations. The remnants of the drum membrane were irregular in shape and covered with blood. In the large perforation I found one larva, which I removed with the ear forceps. This larva moved sluggishly. The malleus was in situ. The Shrapnell membrane was red, covered with blood, but no perforation. A strip of gauze moistened in the solution of liquor aluminum acetate was introduced and bandage applied. Next day the boy was very gay, stating that his ear was now quite well, but that he still has some pain in the ear at times. Temperature normal. No pain over the mastoid. After a week the perforation became smaller and two weeks later was replaced by a scar.

Obviously we had to deal here with absorption of pus from the drum cavity and antrum, caused by the presence of larvæ in the external ear. The larvæ were carried by a sarcophaga fly directly into the ear when the boy slept in the open on a sunny hot day. The chronic otitis after measles has, as we know, a tendency to be cured when proper treatment is applied. I think that the irritation of the edges of the perforated drum membrane caused by scarification accelerated the healing process.

The larvæ I sent for examination to Prof. Z. Mokshetsky (Mokrzecki), a Polish entomologist, the founder and director of the Crimean Museum of Natural History in Simferopol. He recognized them at once and was very pleased to find two larvæ still alive, from which he got cocoons and flies.

The same month I observed the second case, a Tartar girl, three years of age, with larvæ in both ears. The child slept in the open. Purulent discharge from both ears. I removed altogether 13 larvæ, with this difference, that I syringed the external ear channels with 2 per cent watery solution of chloroform. Chloroform, even in so weak a solution, kills the larvae very quickly, and is quite comfortable for the little patient. When applied per se it causes pain by burning the skin. In this case both drum membranes and ossicles were destroyed. After the removal of larvæ the child, who was yelling, crying, etc., as in the first case, became quiet at once. I saw her no more.

Next year (1917) I had three similar cases. All of them were children, and in all the chronic otorrhea was present. After removal of larvæ all these three cases recovered, although in one case the rise of temperature and weak, accelerated pulse, lasted for several days. In no case was an operation on the mastoid indicated, although in all I noticed a tenderness in the region of fossa mastoidea and a rise of temperature.

Among the cases cited by Portchinsky, we find two which ended fatally. In one case (larvæ in the ear, l. c. page 51) a boy 13 years of age, who had had otorrhea for a long time, slept outdoors and was infected by sarcophaga. Four days later, after terrible pains in the ear and in the head, the ear was syringed with milk and turpentine oil, and 30 larvæ came out. Nevertheless, the general condition of the boy became worse, and on the seventh day the boy died with symptoms of meningitis.

The second fatal case was that of a girl, 11 years of age, who suffered from ozena. After sleeping outdoors the child noticed discomfort in the nose, afterwards itching and pain, which grew worse every hour. Some days later a large quantity of stinking fluid escaped from the nose. After eight days of intense suffering the child died from meningitis. The postmortem examination revealed large destruction in nasal cavities, which communicated with orbits. The eye muscles were destroyed. In the nasal cavities altogether 58 larvæ were found.

When we bear in mind that the sarcophaga fly produces, according to Portchinsky, 124 to 168 larvæ, which are very hardy and active, there should be no wonder that under favorable circumstances they may endanger the ilfe of the victim. When, for instance, a dehiscence in the roof of the tympanum and antrum is present it is quite possible that in the absence of the drum membrane the larvæ can penetrate through the dura into the cranial cavity, causing meningitis directly. On the other hand, the larvæ may evoke the absorption of pus by blocking the external ear channel and thus cause an intracranial complication, if not removed at the right time. The best method for removal of larvæ is syringing of the ear with 2 per cent watery solution of chloroform.

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2. Diseases of the Nose, Throat and Ear, p. 652. Ballenger writes about the Texas screw-worm fly (Compsomyia Lucilla macellaria), that this insect can be very dangerous to the life of his victim when the "worm" has penetrated through the bone into the cranial cavity, invading an ear affected with otorrhea.

3. Bezold: Lehrbuch der Ohrenheilkunde, 1906, p. 116. "In der Lehrbuechern finden sich auch durchgaengig Fliegenlarven erwaehnt, welche neben foetider Otorrhoe in knoechernen Gehoergang und der offenliegenden Paukenhoehle zur Entwickelung gekommen sein sollen. Da ein derartiges Vorkommnis weder von anderen neueren Autoren noch jemals von mir gesehen worden ist, so ist wohl ein Zweifel darueber erlaubt, ob hier nicht Taueschungen vorgelegen haben, welche durch raschen Wechsel von Lichtreflexen in der Tiefe entstanden sind."

4. Wohlfahrt: De vermibus per nares excretis Nova acta phys. Akad. Caes. Leop., 1770, p. 277.

5. Portchinsky, I. A.: Wohlfahrtia magnifica Schiner sive Sarcophaga resp. Sarcophila Wohlfahrtii Petersbourg, 1916 (in Russian language).

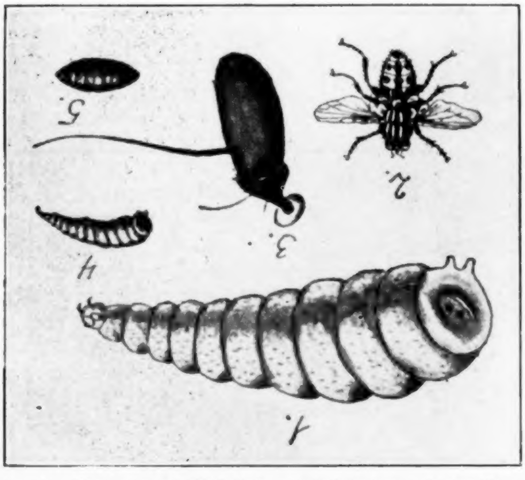

Fig. 1. The Fly of Wohlfahrt (Wohlfahrtia Magnifica).
1. The larva (magnified). 2. The fly. 3. Antenna. 4. The larva (natural size). 5. The false cocoon. (After Wohlfahrt.)

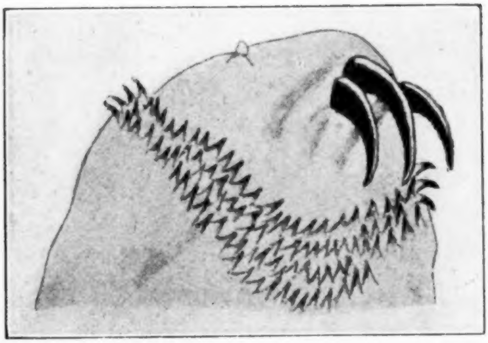

Fig. 2. The head end of the larva, magnified (after Portchinsky).

XXIV.

ADENOCARCINOMA OF THE MIDDLE TURBINATE, ANTRUM AND ETHMOIDS TWO YEARS AFTER RADICAL OPERATION WITHOUT RECURRENCE.

By David N. Husik, M. D.,

Philadelphia.

A. W., 32, white, machinist, married, was referred to the University Hospital dispensary on account of nasal obstruction and pain in the head and face.

H. P. I.: Patient began to complain of frontal headaches in 1915. These headaches would last about 24 hours and would recur almost every week. He gradually became accustomed to them. In January, 1919, he began to notice left sided nasal obstruction and intermittent pain in his cheek, which gradually became worse, and in May, 1919, presented himself for examination.

Nasal examination shows the left nostril completely filled by a large irregular mass, evidently attached to the middle turbinate, boggy to touch, covered with a mucopurulent discharge, and anatomic landmarks could not be easily made out. Cocain and adrenalin had little effect on this mass. Transillumination showed a dark area below the left eye and over two-thirds of the internal aspect of the left antrum. Right side negative.

Under 10 per cent cocain in adrenalin I removed some of this mass, which I at first thought to be polypoid in character. Hemorrhage was quite profuse. The anterior nares was packed and the patient was sent home. On subsequent visits we began using snare and biting forceps, but the bleeding was so profuse that little tissue at a time could be removed. After a half dozen sittings we were able to clear the lower part of the nose, and the patient felt so much better that he failed to appear for further treatment. One morning, on November 2, after washing his face he suddenly had a severe left sided

nasal hemorrhage and was rushed to the hospital. His nose was packed, and after several hours the patient went home. He now complains of severe pain in antral region and left ear, and says he is getting deaf. External examination shows left eye and cheek more prominent. Internal examination shows lower half still free, but the mass has traveled to the nasopharynx. At this time a small piece from the middle turbinate region was removed and sent to Dr. Allan J. Smith, professor of pathology in the University of Pennsylvania, and he sent me the following report:

"Sections of small fragments submitted show a loose fibrous covering of the ordinary appearance of nasal mucosa, at places with persistence of a stratified columnar epithelial surface, but in the main denuded of such layer. Beneath this in loculi of varying size, separated from each other by fibrous septa, is a glandlike tissue with central spaces filled by a hematexylin stained mucin. The lining cells of the acinuslike subdivisions of the larger lobules are, however, not of the large, columnar goblet type to be expected in mucous glands, but small cells of the size of an early connective tissue corpuscle, each with a comparatively large deeply staining nucleus, and these cells occur as multiple as well as single lining layers to the spaces. In the fibrous framework of the growth solid cords and isolated islets of the same type of cells are here and there met, indicating an infiltrative tendency in the growth. The vessels are loose in the fibrous framework of the mass, commonly as irregular channels with indefinite walls. The general appearance of the histologic structure would tend to its classification as an infiltrative adenocarcinoma of origin in a mucus secreting gland such as occur in the mucous membrane of the nose, with modification of the cells to the small size above noted. There is one other interpretation open, which, however, the writer is less disposed to consider, viz., that the growth is of endothelial type (endothelial cancer), extending along and lining lymph spaces into which from unknown source mucin has gained access and is retained."

From November 2nd, when I first began to suspect the mass to be malignant, until December 15, the date of his admission to the hospital for radical operation, I used radium in 50 mg. doses directly applied to the mass with slight improve-

ment. These treatments were given at about seven day intervals, and the capsule was retained from three to four hours. Altogether he has had twenty hours' radium treatment. During this time he was given two X-ray treatments by Dr Henry K. Pancoast.

P. M. H.: Has had the common diseases of childhood; no serious illness; no operations.

F. H.: Mother and father living and well; two brothers and four sisters, all living and well. One sister died of T. B.

P. E.: Patient is a well nourished, well developed, white adult male of good muscular and bony development, but short in stature. No abnormalities in gait and station. Skin of good texture; no jaundice or edema. Head normal in size and shape; scalp negative. Eyes: Left eye shows exophthalmus, pupils equal and react to light and accommodation; no nystagmus, ptosis or extraocular palsies. Ears: Negative to external examination, but complains of pain in left ear and deafness. Mouth and teeth negative. Neck: No cervical or thyroid enlargement. Chest: Well formed and expansion good. Abdomen and extremities negative. Radial pulse regular, volume and tension good. Blood pressure, 133—75. Urine examination, December 18, 1919, practically negative outside of a faint trace of albumen. X-ray report by Dr. Henry K. Pancoast shows disease of left antrum, anterior, and posterior ethmoid cells.

Operation, Drs. Muller, Kornblum and Husik: Intratracheal anaesthesia. Ligation of external carotid. An incision was made in median upper lip to nose and around left side of nose to root, and straight back below the lower lid. The skin and soft parts were retracted, the whole superior maxilla was sawed through. The eye was not disturbed, as the tissues underneath looked healthy. The middle turbinate was completely removed and ethmoid cells broken down to the sphenoid. There was a large mass of grayish jelly substance in the antrum, which was removed, and, together with the maxilla, was sent to the laboratory for examination. The wound was cauterized, packed and sewed up.

December 19, 1919. Patient feels very miserable; complains of severe sore throat and inability to swallow anything.

December 20, 1919. Very miserable; is given liquids through a tube.

December 25, 1919. Stitches removed from face and neck. Patient is somewhat easier.

December 26, 1919. Complains of severe pain in left ear; drum injected but landmarks not obliterated; complains bitterly about his throat.

December 27, 1919. Developed an acute purulent otitis media. Patient was very uncomfortable until January 5th, when he began to show signs of improvement. He was able to swallow some liquid and said his throat felt somewhat easier.

January 7 and 12. Received X-ray treatments.

Discharged from the hospital on the 14th of January in fairly good shape and reported every other day for treatment to throat and ear. Ear became dry on the 25th, and he is able to swallow some soft diet. There is a good deal of puffiness under the left eye, but this seems to be rather a common condition after such operations.

In March of 1920 I saw a little mass in the region of the superior turbinate, which looked polypoid, and after two applications of radium, 50 mg. four hours each, it disappeared, and to date has not recurred. As time went on, the patient improved physically, was free of all pain, was following the occupation of gardener, could chew fairly well, but had difficulty with solid food.

The report of upper jaw and soft parts sent to Dr. A. J. Smith is as follows:

"Portions of the growth developing en masse in the maxillary sinus came into the general laboratory after the excision of the jaw, the latter being submitted to the laboratory of surgical pathology (Surg. Path., No. 8635), whence a diagnosis of giant cell sarcoma was returned. The writer has examined the latter slides and feels that the diganosis given is not justified, but that the sections obtained in the Surgical Pathology Laboratory show essentially the same features as those here about to be described. They were not made of the bone itself but also from the soft tumor tissue found in the antrum. It is learned that the above diagnosis has been changed.

"The sections here examined included in parts only the

intrasinus surface, at places showing a relatively normal stratified columnar ciliated epithelial layer; at other places this is replaced by a single layer of flat epithelium, and at places the surface apparently is completely denuded. Immediately beneath this covering the superficial mucosa is loosely fibrous, more or less edematous, infiltrated by mononuclear and some polynuclear leucocytes, and showing occasional dilated blood capillaries and blood spaces. Beneath this the tumor appears, showing considerable variations on superficial examination but in reality of the same nature generally, with the different appearances occasioned merely by factors of secondary importance. The growth is essentially an adenocarcinoma of the mucous glands of the mucous membrane. At places the glandular growth, while massive, presents well the general structure of glands, and is rather adenomatous than cancerous, no direct infiltration of the interacinous and interlobular fibrous stroma by the epithelium. Here as a rule the stroma is relatively scant and of ordinary fibrocellular character and without special vascularity; but as a rule the epithelium within the basement membrane is excessive and layered. Often, however, a lumen exists containing mucin (sometimes staining normally blue with hematoxylin, but often showing a tendency to take up the pink of the eosin). Where in some portions of the sections the stroma becomes more extensive, excellent epithelial infiltration, cords and islands of definite cancerous arrangements can be found, many fields being rich in this type of growth. Even here, in some of the cords and islets, mucinous collections occur in lumenlike spaces. Elsewhere the stroma shows evident infiltration by homogeneous, eosin staining material, interpreted as probably altered mucin, and at times large areas of such material exist. Into this latter extend lines of epithelium, forming a more or less definite network, the cells at places showing their epithelial character clearly, but in much of such fields being atrophic and resembling more small connective tissue and lymphoid cells. Frequently where the stroma is prominent (as apparently towards the basal attachment of the bits sectioned) there is a rich teleangiectatic plexus of vessels and at places small areas of hemorrhage. In portions showing the frank cancerous character of the growth the epithelial infiltrative cords

show syncytiallike masses of the epithelial cells, these being probably expressions of the rapid local growth of the neoplastic tissue, and these probably gave rise to the appearances interpreted as giant cells of a giant cell sarcoma in the Laboratory of Surgical Pathology."

In the spring of 1921, I referred the patient to the Evans Dental School of University of Pennsylvania for an obturator, which Dr. Fox very skillfully made, and which the patient now wears with no discomfort and is able to chew beefsteak.

My last examination, December 27, 1921, shows the patient still free of any recurrence, nose and throat free of crusts; is working, happy and content with life.

Lee M. Hurd in ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, June, 1918, says adenocarcinoma of the nose is rare enough to warrant reporting each case.

Prognosis: In the opinion of the authorities on this subject, the outlook is most hopeless. Thus Carter states: "Prognosis is bad; only recently Bloodgood has stated that not one of the eighteen cases with malignant disease of the nose operated upon at the Johns Hopkins Hospital recovered. There is no authentic case on record where recurrence has not taken place after the operation and often this recurrence has taken place before the patient has left the hospital."

L. D. Alexander, reporting a case of adenocarcinoma with chronologic report in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, March, 1914, says that adenocarcinoma is essentially a disease of the cancerous age, but an early age is possible, as in the two cases he reported, one of 22 and one of 33. My case was 32 years of age.

His case was radically operated upon and no recurrence after six months.

According to Jonathan Wright, adenocarcinoma is the most frequent malignancy of the nose.

Lee M. Hurd, reporting four cases of adenocarcinoma, says case 1 recurred two years and case 3 recurred one year after most extensive radical operation.

Wm. H. Dudley reports a case of adenocarcinoma, with exophthalmos, which lived two years and five months after a radical operation, including the removal of the eye.

Louis Klemptner reports a case in a female 33 years of age, of possible origin in antrum, and extending to ethmoids and middle turbinate, with no recurrence six months after a Denker and ethmoid removal.

L. C. Minor reports a case in a female of 28 years, origin of middle turbinate, after repeated removal and curetage, with no recurrence after one year. Another case in a female of 22 years with no recurrence after exenteration of middle turbinate and ethmoids.

John McCoy reports a case of origin in ethmoid, with extension to sphenoid and frontal, with no recurrence one and a half years after curetage and modified Killian.

As soon as a diagnosis of adenocarcinoma is made by the microscope (clinically it is impossible, because in the beginning it nearly always begins as polyps or polypoid degeneration of the middle turbinate), the best chance for the patient is complete removal, preferably by the external route, and followed up by X-ray and radium.

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XXV.

CHRONIC STENOSIS OF LARYNX AND TRACHEA WITH REPORT OF A CASE TREATED BY LARYNGOSTOMY AND DILATATION.*

By Edward Cecil Sewall, A. B., M. D.,

San Francisco.

Chronic stenosis of larynx and trachea may be divided into functional and organic types.

Chronic functional stenosis occurs in the larynx. It may be caused by panic or by spasm.

Where fear alone is the cause of the dyspnea, the patient is usually the wearer of a tracheal canula. There is nothing pathologic except the psychic state. It is quite common to have difficulty in removing the canula in the inured canula carrier.

Where spasm is the cause, it produces dyspnea by the median position of the cords. It may be caused by the wearing of an intubation tube. Also inflammatory conditions of neighboring parts may produce it.

Chronic organic stenosis may occur in the larynx, trachea or bronchi. Under this rubric is included:

1. Narrowing of the lumen by pressure from without.
2. Paralysis of the abductors.
3. Ankylosis of the arytenoids.
4. Neoplasms.
5. Hyperplastic and cicatricial lesions.

1. Narrowing of Air Passages From Extraneous Pressure. —Such stenosis is of very great importance, chiefly in connection with goiter, and occurs usually in the trachea. In countries where goiter is common, serious involvement of the trachea is found so frequently that an indirect or direct examination of the trachea is properly asked of the laryngologist in all cases before operation. Death from asphyxiation dur-

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ing the operation has been not infrequent in cases where structural parts of the trachea have been caused to atrophy.

2. Paralysis of the abductors is often the beginnnig stage in recurrent laryngeal paralysis. Following Semon's law, the cricoarytenoideus posticus is affected before the lateralis. The paralysis often accompanies tabes, diphtheria, bulbar and other central nervous system disorders. The cords incapable of abduction are drawn toward the middle line by the adductors. Where both sides are affected breath is passed only with difficulty and noisy vibration of the cords accompanies the effort. The condition is a dangerous one and may lead to sudden asphyxiation.

3. Ankylosis of the cricoarytenoid articulations produces a condition that is distinguished with difficulty from that caused by abductor paralysis. It is caused by "rheumatism," or more commonly by perichondritis. As a result there may be actual joint changes or scar formations about the articulation having the same effect. As in bilateral abductor paralysis cords are fixed in the middle line and thus cause stenosis.

Sargnon and Taubert have called the stenoses resulting from median position of the cords "stenoses fonctionelles." Criticism of this nomenclature by Citelli would seem to be well taken, although they clearly distinguish them from psychic conditions. Citelli calls them "stenoses due to the permanent median position of the cords." Excepting for the position of the cords the larynx appears normal.

4. Neoplasms are provocative of obstruction of the air passages, but in general the treatment is the removal of the growth and is outside the scope of this paper. However, recurring multiple papillomata in their nature lead to chronic stenosis and must be considered. They are much less important from this standpoint than formerly. With direct removal, either through the laryngoscope or with the suspension apparatus, they are successfully combated. Radium seems to have a very definite place in the prevention of recurrence.

5. Hyperplastic and cicatricial chronic stenosis are terms describing with fair accuracy a various group of lesions. The causes of such stenoses are diseases or injuries that lead to hyperplastic or cicatricial conditions blocking the air passages. Among these may be mentioned syphilis, tuberculosis,

scleroma, diphtheria, scarlatina, measles, typhoid, whooping cough, and last, and of great importance, trauma.

Injury may cause perichondritis, inflammation and swelling of structural or soft parts. Killian in his report of war wounds of the larynx divides injuries into those of, first, the parts at the entrance to the larynx; second, above the cords; third, in the middle, through or involving the cords; fourth, underneath the cords. A practical point he brings out in this classification is the fact that emphysema of the skin and surrounding parts occurs only in the wounds of the fourth variety. In such cases the cords form a barrier and the air is forced out of the wound.

Decubitus from an improperly fitting intubation tube or the long wearing of a tracheal canula is considered under the head of trauma. It is an interesting observation that in the inflammatory condition associated with diphtheria, laryngeal stenosis occurs where tracheotomy has been done without intubation almost as frequently as where the patient has been intubated. A frequent cause of the stenosis following diphtheria was first described by Köhl as a hypertrophic inflammatory condition of the subglottic mucous membrane.

TREATMENT.

Functional stenosis, when either simple panic or with added spasm, must be treated primarily along educational lines to rid the patient, usually a child, of fear. The long use of a tracheotomy tube makes the patient more or less dependent upon the consequent free breathing. Breathing through a wide canula bears the same relation to breathing through the mouth that breathing through the mouth does to breathing through the nose. We well know the difficulty in teaching some children to be satisfied with nasal breathing after the free, shallow and easy mouth breathing.

The intubation tube may have led to spasm through undue pressure, and it is recommended to try different forms of tube, more narrow in the interglottic region. Tracheotomy will usually relieve the spasm in a short time, where other means have failed.

Narrowing of passages from extraneous pressure. In such cases the treatment has more to do with the alleviation of the condition pressing on the trachea.

Paralysis of the abductors and ankylosis of the cricoarytenoid articulations give rise to fixation, in the middle line, of otherwise normal cords in an otherwise normal larynx. The treatment of both is identical.

Section of the recurrent laryngeal nerve has been done to produce total paralysis and so leave the cords in the cadaveric position and do away with adduction. There is usually room to breathe in bilateral total recurrent paralysis. Both section and resection of the nerve have not given satisfactory results. It was long ago pointed out by Cain that where there was continued action of the adductors without opposition of the abductors that the cords shortened and so retained their median position, even after severance of the recurrent laryngeal nerve.

Intermittent dilatation by sounds has not been satisfactory nor has the sustained pressure of the intubation tube been sufficient to hold the cords permanently aside. O'Dwyer suggested and tried intubation. He was also the first to cut away the cords for abductor paralysis. Hope also tried excision of the cords for this condition. However, Citelli gave impetus to the method when he excised the cords, experimentally, in dogs, especially to determine whether they regenerated as was claimed by Fränkel. Their controversy brought considerable publicity to the subject. Citelli maintained that the cords do not regenerate, though admitting the establishment of fibrous bands for phonation.

Fränkel gave authority to the regeneration theory, and those opposed to Citelli's method maintained that the voice was lost and the cords returned. The truth seems to lie in the middle ground. Certainly new tissue takes the place of the removed cords. It, however, does not take up the amount of room that the cords did. Also the voice is not destroyed, but, though hoarse in quality, is serviceable. So the procedure has a definite place in the treatment. Jackson has reported cases where he removed the cords, eviscerated the larynx through the mouth, with successful outcome.

The removal of cords alone, however, has not been the answer to the question in most hands, and other means of gaining room have been used. Ivanhoff proposed and carried out submucous resection of the arytenoids following laryngofissure. This allowed the cords to be drawn outward

and gained room for breathing. Resection of the arytenoids has long been done on horses where paralysis of the recurrent laryngeal nerve has allowed them to become flaccid and be drawn into the larynx on inspiration, causing "roaring."

Sargnon and Taubert added the curettage of the sinuses of Morgagni. The adhesion of the walls, they claimed, would draw the cords outward. It is easy to agree with Citelli that the relief was rather problematical and left much to be desired theoretically and practically. Hansberg and Uffenorde removed the soft parts submucously and practically.

Killian developed the technic as follows: After laryngofissure he dissected up the mucous membrane with underlying elastic tissue, conus elasticus, including that in the subglottic space. Removed the musculus vocalis, also the ventricularis and part of the lateralis. Separated more or less of the arytenoid cartilages. Sewed in place the mucous membranes. Fastened the edges of the laryngofissure wound with the skin, forming a laryngostomy.

Treatment of Hyperplastic and Cicatricial Stenosis.—The remedies that have been proposed and used are numerous and diverse. The severity of the stenosis will suggest the radicality of the methods for relief. In therapeutics the number of remedies is in proportion to the difficulties of the task. The various means for relief of these conditions, however, group themselves under three heads: bloodless or dilating, surgical or cutting, and combinations of the two methods.

The first attempts at relief were made by simple dilatation. This treatment was intermittent and was best exemplified in the use of Schrötter's dilating bougies. These were passed in increasing sizes from the mouth through the larynx. It is almost essential that the larynx be not entirely occluded. Where it requires force to pass the smallest sound grave damage may be done by the formation of a false passage. Cutting through an impermeable stricture from above, except in web formation or where the knife is guided with great certainty under the eye, is dangerous. This intermittent method of dilating has been satisfactory in some cases and is still useful where the stricture is a mild one, easily discouraged.

O'Dwyer with his intubation tubes brought a method of great value which has had tremendous influence upon all the

various procedures, surgical and otherwise, used today. While intended, originally, for acute stenosis of the larynx, O'Dwyer soon saw the usefulness of his tubes in chronic conditions. His great skill and the ingenuity of his successors, especially in the American school, have sufficed to conquer by intubation alone unbelievably difficult stenoses. Intubation may be preceded by intermittent dilatation or the use of the expanding dilator until the passages will admit the smallest tube. By their use in successively increasing sizes and with variations as to composition was obtained the ultimate advantage from sustained dilatation, by way of the mouth.

Tracheotomy is considered by this school a disadvantage and like the other operative procedures is only to be employed where intubation has failed in its purpose. Where tracheotomy has been performed greater variety is permitted in the methods of dilatation. It is now possible to dilate the stricture from below. Urethral catheters, or better, the dilators devised by Dr. Lynah, serve this purpose. This intermittent bougieing would correspond to the use of the Schrötter type of sound by mouth. Uchermann has combined the incision of the stricture from below with blunt curved bistoury where necessary before dilatation. This is an easier and safer use of the knife than from above downward.

Making use of the tracheotomy wound, it is now possible to draw down, by means of the "endless cord," different forms of dilating apparatus. The intubation tube may be anchored through the tracheal fistula by some form of "post." Care should be taken that the post and the tube are not both of metal, because corrosion may fix the post and make removal difficult. If the tracheal canula is in situ combinations of all kinds may be made with variations of the obturating part of the apparatus. An intubation tube, one covered with rubber where desirable on account of size or composition, a single piece of rubber tubing, a piece of rubber tubing twice the requisite length doubled upon itself—any one of these suitable to the case may be drawn down into the larynx and fastened by cords to the tracheotomy tube or about the neck. A further development of this same principle is represented by the different forms of tracheotomy tube with dilating plug attached to it by some mechanical device. These tubes have been de-

veloped so that they can be separated into units small enough for ready introduction and then brought into position and locked.

Where all the simpler methods fail or where, in one's judgment, they are inadequate, there is fortunately a method which will satisfy reasonable requirements of breathing and phonating in practically every case. Laryngostomy as at present employed is the result of the development of the idea of Herying, worked out principally through the Lyon school, as represented by Sargnon and with the essential aid of Killian.

The surgical details being given in extenso by Jackson, Sargnon, Killian, Moure and others, we will simply sketch the salient features. Briefly the operation consists of

1. Laying Open the Larynx and Trachea.—Jackson's idea of inserting scissors or cutting instruments into the tracheal wound and cutting through all structures, including the skin, in one motion is a great time saver and also favors the wound epithelialization. It leaves a wound edge with successive layers undisturbed by dissection. Where the tracheal wound is lower than the stoma desired, or where irregularly placed, as reported in war wounds particularly, there may be advantage in careful dissection of skin and successive tissue layers.

The excision of the intralaryngeal parts where the stenosis is of the median cord position type has been discussed.

How much of the scar tissue to remove must be determined by the operator in each case. What can be done submucously is certainly advantageous, but the difficulty of the submucous resection of any old inflammatory tissue is familiar to rhinologists. Most cases can be successfully combated by dilatation. Jackson recommends a cut straight through the scar tissue in midline, care being taken not to encroach upon the esophagus.

2. Dilatation of the Cavity and Establishment of Stoma.—Killian's valuable contribution to the operation was the use of soft rubber tubing in increasing sizes for the purpose of dilatation. He showed that the constant pressure of the soft rubber accomplished the purpose with minimum tissue necrosis.

Some tissues are more resistant than others, and the practice is to remove the tubes, watch for inflammatory signs and later replace, gradually increasing sizes of tubes.

Many forms of tube have been used. The combination, whatever it may be, must furnish air as well as pressure. There are three different principles on which the tubes have been built:

First, a simple tracheotomy tube with rubber dilating plug extending upward from its dorsum through the larynx to the level of the base of the epiglottis. This rubber plug or tube is fastened in some manner to the tracheotomy tube. Breathing is through tracheotomy tube, and the patient is unable to phonate during treatment.

Second, a straight hollow tube laid in the cavity and extending from trachea up through the larynx as before. Here the patient can breathe only through the rubber tube, but can phonate during treatment.

Third, Jackson's combination of a tracheotomy tube and the upright rubber tube. The tracheotomy tube has a straight post vertically placed, on the upper end of which is fastened the rubber tube extending upward as in other cases. The added feature of a curved tracheotomy tube connecting through the wound with this straight tube gives the patient ample breathing space and, on closing the external tube with the finger, allows him to phonate.

3. Closure of Stoma.—It is best not to close the external wound until all possibility of further contracture is minimized. The wound causes but little inconvenience, more than offset by the sense of security enjoyed by the patient. However, there is a certain added danger of lung infection from its presence, and so it should be closed as soon as advisable. All cases with tracheal fistulæ have been found to have chronic inflammation of the trachea, often of extreme degree.

If the wound has become in time very narrow, the edges may be freshened and closed by simple suture. Usually the gap is such that some form of plastic is necessary. Again, if not too wide, the turning over of skin flaps will suffice, first with raw surface out and the next drawn over this. There are a number of different plastic methods of value.

Where the stoma is very wide, however, there may not be enough structural value in the skin alone. In such cases transplantation of bone or cartilage into the flap is necessary, ac-

cording to the methods of Konig, Mangold, Schimmelbusch and others.

STATISTICAL RESULTS OF LARYNGOSTOMY.

Sargnon reviewed 150 cases, 76 per cent cured, 9½ per cent no result, 8½ death. Of his own personal cases, numbering 18, he reports 3 deaths, or 16.6 per cent.

St. Clair Thompson, 36 cases, 6 deaths, 16.6 per cent. Ferreri, 11 cases, all cured, no deaths. Chiari, 20 cases, 7 cured; 2 left his service; the rest still under treatment; no deaths. Jackson, 1915, 18 cases; ultimate recovery in all cases. Bellancioni, 16 cases. Schnabel, 2 cases; war injuries. Joseph Beck, 1915, 3 cases with excellent results; no deaths.

Killian, 1916, 36 cases war injuries; 25 fully cured, 8 still under treatment; good breathing space and more than useful voice; no deaths.

Moure, 1918, 24 cases; 9 cured, 15 under treatment. Brunnings, 1918, 6 cases, all under treatment. Hofer, 1920, 11 cases; favorable results; no deaths.

The statistics show that in recent years the number of deaths from this procedure is almost nil. Also the voice in nearly all cases has been reported as useful, and in many instances excellent. It is usually of hoarse quality and explosive character.

Iwanoff made tracings of the movements of the air from the mouth in normal individuals and patients cured by laryngostomy with dilatation. The normal curve shows a sharp rise during inspiration with but little change in the long sustained curve during phonation.

Those operated upon showed the sharp rise for the inspiration, a short sustained period with another quickly following inspiratory effort. Normal speech requires very little breath, but these patients get up pressure and use it quickly.

The time of treatment varies from a few months, in exceptional cases, to two years or more. The average time of Moure's cases was about one and one-half years.

The following case has been under the care of myself and associate, Dr. J. A. Bacher, who has had equal share in its management:

Referred by Letterman General Hospital, March, 1919. Private A. D. Was struck by shrapnel October 10, 1918.

Projectile entered right side of neck about the level of hyoid bone, passed downward and inward directly through the larynx, making exit on left side below the level of the thyroid cartilage. Tracheotomy performed in France. Has worn tracheotomy tube ever since.

Status praesens: Patient can force air through larynx and so produce strained weak sounds. Can swallow without difficulty, though immediately after injury food passed out through wound. Epiglottis is bent upon itself, displaced laterally and bent over larynx. The structures of the larynx are unrecognizable, either with laryngeal mirror, direct laryngoscope or suspension. Could pass a small probe, two millimeters in diameter, into the trachea.

April 23, 1919. Cocainized larynx and trachea through the tracheal wound, anesthetized skin and deeper parts with one-fourth per cent novocain. Introduced scissors through the tracheal wound, cut through the skin, thyroid and cricoid cartilages, making the tracheal opening continue with the opening into the larynx. Retracted the edge of the wound, being careful not to injure the cartilage.

The structures inside the larynx were unrecognizable. On account of the adhesion it was difficult to hold the walls of the thyroid apart very far. Cut directly backward as near the esophagus as deemed safe. Did not attempt to remove any of the redundant tissue.

Introduced the Jackson laryngostomy tube, number six, and brought down on top of it a piece of rubber tubing, 15 millimeters in diameter, two pieces telescoped, so quite firm, inside diameter six millimeters. This was tied by silk threads to the laryngostomy tube, the upper end at level of top of arytenoids. The wound was then very firmly packed so as to remain open.

Patient was placed in semireclining position in bed and the dressing moistened with bichlorid, frequently, changed by day, and by night, nurses. He had a little difficulty with liquids at first but very soon was handling his food quite easily. He also soon became used to the discomfort of the tube. He left the hospital May 10, 1919. The subsequent history of the case, though tedious, was uneventful.

The wound was held open by gauze until the establishment of the stoma. Laryngostomy tube was removed first, after

about two months. There was no evidence, during this time, of any inflammatory trouble from the tube. It was changed again in one month.

September 10, 1919. The laryngostomy tube was taken out, left out for three days and then an extra large bronze intubation tube was inserted through the mouth and fastened in place by a plug, through the tracheotomy wound. The patient at this time might have been considered cured, as the breathing was good, voice fair, and the larynx epithelialized. The remaining treatment has been to guarantee against constriction.

November, 1920. Up to this time patient wore the large bronze intubation tube with post, a month at a time, every other month. Then the tube was permanently removed and the patient was taught "autobougieing." A rubber veterinary catheter 15 millimeters in diameter was cut off at convenient length, passed over a heavy piece of tightly fitting copper wire and bent at a rounded angle. The rounded end was thus easily passed into the trachea through the larynx and, after a few days of instruction, the patient learned to pass the dilator and keep it in place a considerable time.

We advised him to use the dilator daily or at frequent intervals for an indefinite period, as it is quickly done with little inconvenience.

June, 1921. Believing him now safe from recurrence, we closed the stoma. The wound had gradually narrowed so that the skin was simply drawn over the opening, the edges freshened and sutured The closure was complete and satisfactory. There was very little space for raw surface to cause granulation, but if such should form we rely on "autobougieing" to take care of it.

The patient has a very useful voice, produced by vibration of the soft parts at the entrance to the larynx. These tissues can be seen to vibrate on phonation. He can make himself heard at long distances. We tested him at 100 yards and could distinguish his words very readily. His voice has the usual explosive quality. Since leaving our care the patient has married.

Our experience has been limited to this one case and another which is at present in the course of treatment. I pre-

sume that always in such cases individual resource leads to some modification of standard methods. We did not follow out the dilatation as advised by some surgeons, especially Moure. He establishes the stoma, and then after that has healed begins his dilatation.

Our case took from the start a sufficiently large tube, and it was not changed for two months. This may not be safe in all cases, but certainly caused no harm in this instance, and we are following the same procedure in the other case which we are treating.

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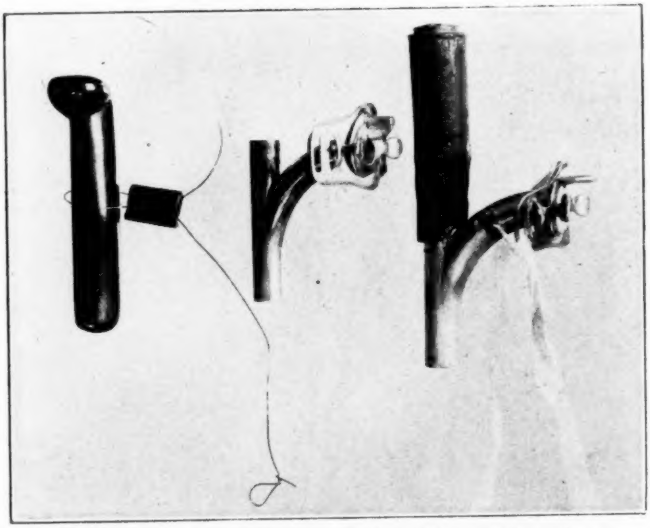
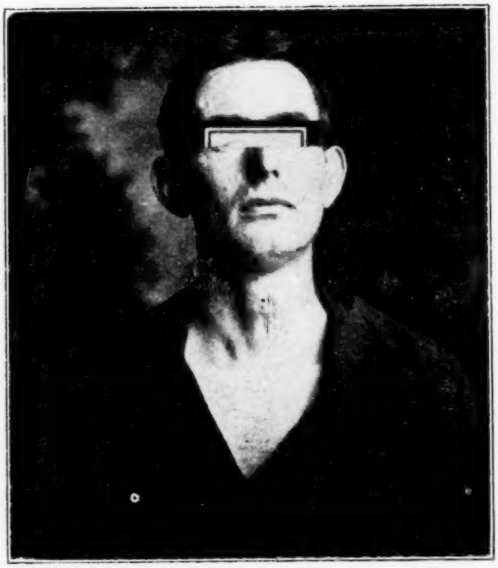


Jackson's tubes and intubation tube with retaining wire.

Patient showing stoma established.



Patient showing wound some months after closure.

XXVI.

THE HISTOPATHOLOGY AND HISTOGENESIS OF BENIGN GROWTHS OF THE NOSE AND ACCESSORY CAVITIES.*

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It has been the custom of the author of this paper to give more than average attention to the science of pathology in its bearing on otolaryngology, and he believes that this study of a branch of medicine somewhat removed from the usual routine of special work has been the source of much valuable information relative to the specialty and of a better understanding of many cases. As a knowledge of anatomy is essential to proper surgical skill and technic, so in a no less definite way is a knowledge of pathology assential to a proper understanding of diagnosis and treatment. We practice a somewhat limited specialty as otolaryngologists, and it is not too much to ask that we have a working knowledge of the fundamental sciences as applied to that specialty. We may not always be capable of making a histologic diagnosis with the accuracy expected of one making pathology his specialty, and it is not with such an expectation that attention is directed to the science, but in the hope that we may gain a clearer understanding of disease processes and of the changes taking place in the tissues with which we work.

The illustrations which accompany this paper have been limited to microphotographs, thus avoiding the tendency to draw into an illustration what we think should be seen in the section rather than what is actually present there. In this connection it must be kept in mind that no photograph can convey the impression gained from a study of the whole section, but a careful selection of the proper area for reproduction has been made in each case. The photographs were made

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by the author, using a very simple apparatus, consisting of an ordinary camera box with bellows and ground glass back and without lens, mounted on a vertical support. A Bausch and Lomb balopticon was used as a source of light, interposing a piece of flash opal glass for diffusion while filtration was secured with Wrattan gelatin filters. Orthochromatic plates were used. Exposures were made by turning on and off the balopticon, thus avoiding the use of a shutter.

The paper makes no particular claim for originality, but is more the result of a careful study of the literature and an endeavor to utilize that study to the best advantage; with an earnest effort to collect a series of specimens typical of the conditions they represent and a careful selection of those proper to be reproduced and presented herewith. Many have been discarded as unworthy, others have been selected in lieu of something more nearly perfect, and still others have perforce been omitted because it has not been the author's good fortune to have met with cases from which to secure specimens.

THE EDEMATOSES.

These, by far the most common pathologic conditions met with in the nose and accessory cavities, have been variously called polypi, edematous polypi, myxomata, fibromyxomata, myxofibromata, soft fibromata and edematous fibromata. Many writers have erroneously adopted terms descriptive of them which have not only resulted in a confusion of terminology but of understanding as to the actual histologic structure of the tissues under consideration. There is nothing in the structure of a nasal polyp which justifies the use of the term myxoma as applied to it, for it contains no myxomatous tissue in its substance. Wright,[49] in 1900, emphasized this point very strongly, and it is also brought out in Delafield and Prudden[16] and by Ewing in his "Neoplastic Diseases,"[18] although many of the older pathologists fail to recognize this differentiation. Expressions such as "fibromyxoma" and "myxomatous" have doubtless been used by many writers with a full knowledge of the laxity of the terms, but they have failed to transmit that knowledge to their readers, with the result that there still exists a deplorable lack of uniformity of under-

standing and of terminology among rhinologists. The writer pleads guilty to this fault in his own case in the past and feels that a more accurate terminology should be adopted, if not histologically descriptive, at least not actually misleading. To this end the general term "edematoses" will be used in this connection, for the reason that a study of the histology of these growths will reveal that the fundamental underlying change in the tissues is a serous effusion, an edema.

SIMPLE EDEMATOSIS.

To that condition in which there is simply an edema, or serous infiltration, among the fibers of the loose areolar tissue constituting the subepithelial layer of the mucosa and unaccompanied by demonstrable fibrous hyperplasia or marked pathologic change in the tissues themselves, the term "simple edematosis" will be applied. This classification should include such conditions as the apparent hypertrophies due to hay fever and socalled vasomotor rhinitis, many of the more lasting polypoid hypertrophies of the turbinates, resulting, usually, from chronic conditions in the sinuses, and some of the earlier types of pedunculated polypi before they have undergone marked fibrous change. Figs. I, II and III.

EDEMATOUS FIBROMA.

On the other hand, it is true that many of the edematoses will reveal a sufficient amount of fibrous hyperplasia to justify the name of fibroma, and to these will be applied the term "edematous fibroma," as most descriptive. By this is meant a tumor consisting of loosely packed fibrous tissue, the site of serous effusion, containing fibrin threads, leucocytes in the form of the familiar round cell infiltration, blood vessels and at times glandular structure. In short, it may contain any of the constituents of the mucosa from which it springs, to which may be added the signs of subacute or chronic inflammation evidenced by increase in round cell infiltration, increased fibrosis around vessels, dilatation of gland acini and in places marked fibrous tissue hyperplasia. The epithelial changes manifest themselves both as hyperplasia of the normal columnar and in the form of metaplastic change to cuboidal or squamous, the latter usually stratified and frequently hyperplastic. Figs. IV, V and VI.

This classification includes the older forms of turbinate and septal edematoses and the majority of those having their origin in the accessory sinuses. At this point it is only fair to remark that the gradation from the purely edematous conditions to those justifying the use of the term edematous fibroma must be very gradual, and that there must of necessity be a certain elasticity in the application of these terms. It is true, however, that at one extreme there exists the typical simple edematosis and at the other the condition of undoubted fibrous hyperplasia with edema, and although histogenetically they are apparently one and the same process, their evolution from one form into the other justifies the use of slightly different terminology to signify that change.

The histogenesis of this type of nasal neoplasm has been deeply studied by numerous authors, among them Uffenorde and Hajek, whose views are quoted by Marquis,[33] Skillern[47] and others, and it is agreed that their formation is undoubtedly dependent upon some form of irritation or inflammation in the tissues from which they spring. This results primarily in transudation of serum and later in hyperplasia of connective tissue with definite tumor formation, necessitating an accompanying hyperplasia of the epithelial covering to keep pace with the increase in substance of its underlying structures.

PSEUDOCYSTS.

A condition not yet mentioned but frequently occurring in the development of edematous fibromata is that of socalled cyst formation. Although true cyst formation does occasionally occur in the mucous membranes of the nose and accessory sinuses as a result of obstruction of the opening of a mucous gland, the socalled cysts found so frequently in the pedunculated growths in the nose are in reality pseudo cystic in character and are simply dilated connective tissue spaces filled with serum. They are not lined with epithelium as are the true cysts, but with endothelium derived from the connective tissue in which they are formed. Figs. VII, VIII, IX and XI.

ANTRAL, CHOANAL OR NASOPHARYNGEAL POLYPI.

The edematoses in general may have their origin from any portion of the mucosa lining the nose or accessory cavities,

but spring most frequently from the ethmoid cells and middle turbinate. When arising from the antrum they have attracted enough attention among rhinologists to have been given a special name, that of antral, choanal or nasopharyngeal polypi. Histologically they are identical with the edematoses as described above, but their peculiar origin and location make them worthy of special consideration in this connection. Since Zuckerkandl in 1893 called attention to their origin they have been a subject of considerable interest, and Woakes, Hajek, St. Clair Thompson and Killian among others have treated the subject thoroughly and have established beyond question the fact or their origin in the antrum. Avellis[4] in 1913 and Ewing[20] in 1918 have reviewed the subject again, and Ewing has taken up that portion of the subject which is germane to this paper. He reports thirty-five cases, of which fifteen were studied histologically, and his observations in this field agree with those of the author[5] expressed in a previously published paper but based on a much smaller number of cases. His conclusions regarding their development differ from mine in one detail, namely, the mode of exit of the polyp from the antrum. It has been my conclusion, from a study of the histology and the gross pathology at operation, that these growths start as either an edematous fibroma or as a cyst of the mucosa from a chronic inflammatory process, probably not suppurative. This fills the antrum, and extrusion through the antral opening occurs as a result of pressure. Plausibility is lent to this theory by the fact that at operation the antrum is usually found to be filled by the growth, that studies of the antral mucosa show an older or more advanced stage of pathologic change in the antral tissues, and because this is the only reasonable, logical conclusion that can be entertained. Ewing maintains that it certainly does not occur in this manner, for the reason that "the antral portion has often been found to be quite small, occupying only a small portion of the posterior part of the cavity." Such is sometimes the case, but since histologic observations show that the antral portion of the growth is always of quite marked fluid content, it is quite logical and reasonable to suppose that after its extrusion through the ostium and its resultant free expansion in the nose there occurs a migration of fluid from the antral tissues into the nasal

portion. This would be favored by the greater fibrosis of the older pathologic condition in the antrum and would result in the apparent loss of substance in those tissues. Or, knowing as we do how rapidly under certain conditions it is possible for edematous tissues to shrink from absorption of their fluid contents, is it not logical to suppose that such a simple occurrence might explain the condition in some instances? Figs. X and XI.

PURE FIBROMA.

Although pure fibroma, commonly called hard fibroma or fibroma durum, is exceedingly rare in the nose and accessory cavities, authentic cases have been reported. My search of the literature has revealed but three: one reported by Meigas[37] as fibroma of the septum, but with no histology has been eliminated as having probably been an edematous fibroma, as no other case of pure fibroma of the septum has been reported. Another, not included among the three, is reported by Dabney[15] as having undergone sudden malignant degeneration, which leads to the belief that it was fibrosarcoma from the beginning, as malignant degeneration is questionable in pure fibromata. The original sections in this case may not have reached sarcomatous tissue. Worthington's case,[48] however, appears to be authentic, as is also that of Dempsey,[17] particularly as the latter underwent retrogression and disappeared spontaneously. This growth sprang from the sphenoid and was not limited to the nasal cavities, extending into the nasopharynx as well. Coates' case[13] of multiple fibroma had two parts, the nasal part being separate and distinct from the nasopharyngeal, and for that reason it has been included as being a nasal fibroma. Nasopharyngeal fibromata are more common and although outside the scope of this paper their histology is identical.

Here again we meet the same difficulty in the literature resulting from loose application of descriptive terms, and in view of the fact that most of the benign growths of the nose are, in a sense, fibromata the adoption of a more accurately descriptive term is suggested, that of "pure fibroma."

The histologic structure of pure fibromata consists practically entirely of closely packed fibrous tissue, showing very

few nuclei, blood vessels or cellular forms and none of the characteristics of the nasal mucosa. They may be the seat of hyaline change or of calcification. They have their origin in the periosteal connective tissue, and Zandonini[55] ascribes an etiologic significance to inflammatory processes.

PAPILLARY FIBROMA.

This subject cannot be properly treated without reference to Wright's[50] admirable review of the subject, wherein he took issue with Hopmann and others, and showed beyond question that Virchow's terminology of papillary fibroma should be adopted as the descriptive term for these growths, limiting the term to mean a tumor consisting of a profusely proliferated epithelial layer supported by a very small amount of connective tissue with small, poorly developed blood vessels. The epithelial layer has separating it from the connective tissue a delicate basement membrane, demonstrable in most cases, and a few round cells may be found scattered among its fibers. Mucous glands should be absent from the connective tissue stroma, as the tumor develops from the epithelial and subepithelial layers of the mucosa, which contain no glands.

Careless histologic diagnosis has caused great difficulty in the interpretation of the reports of some observers, for the reason that they have often been led to make a diagnosis of papillary fibroma by the papillated appearance of the mucosa caused by infolding from pressure and without the characteristic epithelial proliferation. It is safe to state, however, that the occurrence of papillary fibromata in the nose is rare and their commonest site is on the septum. Arrowsmith,[3] in 1911, reported the thirty-fifth case up to that time, not including the one of Blumenthal[6] reported in 1910, all of which were septal growths. Herzheimer,[28] in 1911, mentioned one case originating in the frontal sinus, Hannemann[26] one originating in the ethmoid and sphenoid, reported in 1912; Anziolotti[2] one in the same year, Johnston[30] one and Brueggemann[8] two in 1913, Callison,[9] one springing from the region of the bulla and middle turbinate in 1916, and Ide,[29] in 1917, a case in which two were present, one springing from the septum and one from the anterior end of the inferior turbinate. In all of these we are justified in concluding that the histologic diagnosis was

carefully and properly made, and although all were not located on the septum it is fitting that they should be considered and classed together as papillary fibromata, regardless of their point of origin. Figs. V and XII.

Scarlett[44] calls our attention to another question of interest in this connection, that of the socalled hard and soft varieties of papillary fibromata. This nomenclature has followed the rule that if composed of squamous epithelium the tumor is called hard, if composed of columnar epithelium it is called soft. It is obvious that such a method of terminology is purely arbitrary and should form no part of the histologic diagnosis. It should be replaced by the terms "squamous celled" and "columnar celled," and in Herxheimer's paper[28] he has made use of these terms, calling his case one of "fibroepithelioma papillare cylindrocellulare."

ADENOMA AND ADENOFIBROMA.

Here, as in almost all benign nasal growths, one finds in the literature a confusing lack of agreement as to proper terminology. Ewing's observations[19] suggest that the only true nonmalignant adenoma is that owing its origin to embryonal tissue and adenomatous morphology in general is due to various forms of irritation, functional activity or overgrowth. He recognizes, however, that it is not always possible to make the distinction on morphologic data alone without considering the conditions under which the process develops and its clinical course. Wright and Smith[52] also state that "benign adenoma unassociated with marked evidence of its origin in the stages of chronic inflammation of the nasal mucosa is an exceedingly rare phenomenon." The most interesting case encountered by the author in this connection is that reported by Gault,[21] in 1911, in which the growth is described as filling the whole nasal cavity and attached to the external wall at the ethmoid level and to the roof and necessitating the Moure operation for its removal. The histologic diagnosis of benign adenoma was confirmed by Professors Jacques and Hoche of Nancy, and no evidence of recurrence was noted after five months. Caldera[11] in the same year reported a small tumor growing from the inferior turbinate and the meatus, the histology of

which was that of adenoma. These, with Mosher's case[38] and two of Wright's, are the only ones met with by the author that could be classed as benign adenomata, and Mosher, in his article, quotes Hasslauer as reporting but four cases up to 1900. On the other hand, growths partaking of the nature of adenomata but in reality adenofibromata are not so uncommon in the nasal passages. Johnston reports two cases, one in 1909[31] and one in 1913,[32] which he calls fibroadenomata and describes as consisting of masses of fibrous tissue surrounding islands of glands. Allen[1] also reports such a case, which he describes as normal appearing gland tissue embedded in dense fibrous tissue with areas of calcareous degeneration and some areas of new bone formation. Myxomatous degeneration was also evident in places in the fibrous tissue. It is clear that these latter cases fall into the class of inclusions and cannot be considered as true adenomata, being primarily fibrous hyperplasias which include in their mass islands of normal gland structure. Their proper descriptive term, therefore, should be that of "adenofibroma," thus reserving the term "adenoma" for such rare occurrences of this tumor as may present themselves. Fig. XIII.

Wright,[51] in reporting two cases denominated by him benign adenomata and which were practically identical histologically, gives the following description of their appearance microscopically: "Numerous fingerlike processes springing from a common base. These processes are made up of loose edematous fibrous tissue covered by one or two layers of columnar ciliated epithelium; but it is apparent that the epithelial development is a complex one. In no place is there any marked thickening of the layers, but the surface epithelium is seen to communicate and to be continuous with deep indentations and ramifications of it into the underlying stroma. Separate rings of epithelium, ovoid, circular or ramified in shape, and varied in extent, are seen to occupy and make up a large part of the bulk of the growth. Examined with the high power one is immediately struck with the amount of mitotic granules in the epithelial cells. Evidently proliferation is rapidly going on, but not in such a way as to increase markedly the number of layers." This description is so much superior to any of which the author is capable that he quotes it as a substitute, particu-

larly as it has not been his fortune to see sections from a true nasal adenoma.

ANGIOMA AND ANGIOFIBROMA.

These growths, most frequently termed bleeding polypi of the septum, are also subject to much confusion in their terminology, and for several reasons. Primarily, they were thought to be limited to the septum, but Malen[32] in reporting two cases described by him as telangiectatic fibroma and telangiectatic adenomatous fibroma, suggects the term bleeding polypi of the nasal fossæ. Citelli,[12] in reporting five cases, one of which was not situated upon the septum, deals strongly with this part of the subject and advocates discarding the term bleeding polypi of the septum. Matsui[35] reports two cases, both of which he speaks of as bleeding polypi of the inferior turbinate, describing them histologically as consisting of "hyperplasia of connective tissue and increase in vessels, the latter grealty dilated." The cases of Mayer[36] and Callison[10] were both of septal origin, the former defined as a "cavernous angiofibroma" and the latter as a "polypoid hemangioma." Reamer[41] reports one case of angioma of the antrum histologically pronounced benign. Thus it may be seen that the terminology should not include the location, for although most such tumors spring from the septum they may have their origin from other parts of the nose and accessory cavities.

This conclusion, however, does not settle the vexed question of terminology and classification as applied to them. Mosher,[38] referring to Haslauer's statistics, leaves the impression that the preponderance of benign new growths of the nasal cavities are angiomata, as out of 115 growths reported 57 were of that type. Wright and Smith,[53] on the other hand, state that "nasal angioma is a rare form of tumor." This evident disparity is explained by the usual laxity in application of descriptive terms and is probably due to the fact that apparent tumors consisting or simple granulation tissue are comparatively common, especially on the septum. Citelli[12] states: "It will be observed that the etiology of bleeding polypi is the same as that of all tumors, or of any neoformations upon which irritant causes have a marked influence. These irritant causal factors sometimes set up simple or spe-

cific granulations, at other times true tumors. These granulations must not be confused with the veritable tumors." Russo[42] also, in reporting five cases under the title of bleeding polypi of the nose, recognizes this differentiation by diagnosing two of his cases histologically as granulation tissue only. He also refers to Citelli's statement to the effect that with the denomination of "bleeding polypi" tumors of various kinds are united in the same class, as, for instance, the granulations and pure angiomata; and quotes Macaigue, Casalie-Brunetti and Denogany, who seem to think that all socalled bleeding polypi should be united in one group and, taking account of their histologic and clinical characteristics, called "fibrovascular benign tumors of the nose." In spite of all this, Russo clings to the old term, thinking a change would only confuse and would solve the problem for no one, but conceding that among the bleeding polypi there should be included neither the pure angiomata nor the granulation tissue masses.

Citelli, in the paper referred to above, goes on to discuss at some length this part of the subject, proposing that "all nasal tumors be classified according to their histologic structure, following the actual rules of pathology," and after a detailed description of his cases, states: "As a matter of fact, the larger part of the nasal tumors to which the designation "bleeding polypi" has been applied are true angiomas: simple angiomas, cavernoma (or more often mixed, simple and cavernous): or mixed angiomas, with connective or myxomatous tissue." Personally, the author can think of no good, scientific reason why these tumors should not be classified according to their histology as granulation tissue masses, angiofibromata, either simple, cavernous or mixed; and pure angiomata, either simple, cavernous or mixed. The term angiomyxomata would be inapplicable here for reasons previously elaborated.

It seems likely, from a study of the description of many of the cases reported by Citelli, Russo and others, that the term "angioma" has in many instances been applied to tumors more properly falling into the present classification of "angiofibromata." There must, of course, be a certain elasticity in the application of these terms and the personal bias and opinion of the observer must be taken into consideration.

Histologically, the granulation tissue masses are simply collections of rapidly growing blood vessels, their walls formed by a single layer of endothelium and supported by a stroma of young connective tissue containing many cells.

The angiofibromata are made up of blood vessels supported and surrounded by fibrous tissue of varying degrees of density and in some instances the site of edematous infiltration. The vessels, particularly those of capillary size, often consist solely of a single layer of endothelium, although in many of the older tumors with larger vessels the vessel walls may present both fibrous tissue and muscular structure, the latter rarely. The vessels run indiscriminately through the tumor mass so that in sectioning they are cut at various angles. Endothelial proliferation in the extravascular substance is common, particularly in regions showing rapid growth of small vessels, so that in some cases the intervascular structure is largely made up of endothelial outgrowths from the vessel walls. These cells sometimes form themselves into rows as if in an effort to form new vessels. The cavernous types usually show the above picture in some sections, with the addition of dilated blood spaces in others. These spaces frequently show an increase of fibrous tissue in their walls, including proliferating endothelial elements and some fibroblasts. Extravasations of blood into the fibrous tissue stroma are frequently observed. The epithelial covering is usually stratafied and may be either the normal columnar or squamous, the latter usually in the anterior part of the nose from local irritation.

The pure angiomata, practically never met with in the nose, are made up practically entirely of vessels with virtually no fibrous supportive structure and may be either capillary, arterial, venous or cavernous. Their intervascular substance may be made up entirely of endothelial elements and their covering is subject to the same variations as above. Figs. XIV and XV.

ANGIOMYOMA.

Citelli,[12] in the paper referred to above, adds the report of a case of nasal tumor which he defines as an angiomyoma and describes as consisting of capillaries, mainly arterial, which presented a marked hyperplasia of their muscular layer. These vessels were interwoven with and surrounded by bundles of

pliable muscular fibers with characteristic elongated nuclei and such fibers enveloped the surface of the tumor like a capsule. It appears that in this instance there occurred in the muscular coat of the vessels a marked hyperplasia resulting in the above histologic findings and as it is the only case of its kind on record is of corresponding interest.

LYMPHANGIOMA.

This condition is so rare in the nose as to receive no more than passing mention in the literature. Johnston[31] mentions a case reported by A. Schmidt, and Wright and Smith[54] refer to one case reported by Hamm and also to one reported by Hajek and Polyak[27] called lymphangiectasis myxoma but which they suspect was a myxosarcoma.

Histologically, lymphangiomata are tumors composed of lymph vessels instead of blood vessels, as in the hemangiomata and their microscopic appearance is very similar to the latter. Never having seen a lymphangioma of the nose, the author will not attempt a description of one.

PAPILLARY HYPERTROPHY OF THE TURBINATES.

This style of hypertrophy is sometimes so marked as to form what is practically a tumor and will be included in this paper for that reason. Although most common on the posterior end of the inferior turbinate, it has also been observed on the anterior end and on the posterior end of the middle turbinate. Its pathologic interest is the same, regardless of its site, and one description of its histology will suffice for all.

The chief factor in the histology of this condition is the marked hyperplasia of the fibrous tissue in the subepithelial layer, usually unaccompanied by edema. This results, of course, in engorgement of venous spaces, in partial obliteration of glands from pressure with dilatation of some of the acini, and in hyperplasia of the epithelial covering. The latter occurs as a direct result of the papillation of the surface produced by fibrous overgrowth, necessitating epithelial proliferation to keep pace with the increase in surface area. Metaplastic changes are also frequently met with in certain places, accounted for by the irritating effects of pressure and disease upon the epithelium. Fig. XVI.

CHONDROMA.

This type of tumor is also of great rarity in the nose, although many such have been described by authors who have doubtless taken septal spurs (ecchondromata) to be such. Sherrill[40] reports a case of enchondroma of the antrum, and Schwerdtfeger[45] also reports a tumor diagnosed as genuine chondroma of nose, frontal sinus, antrum, ethmoid and sphenoid. His histologic findings are quoted: "The section shows the tumor consisting of several small nodules, connected with little tissue containing few blood vessels. The cartilaginous tissue itself contains numerous cartilage cells and some homogeneous matrix negative to elasticin staining and presenting in spots distinctly fibrillary structure." He concludes that these areas show commencing mucous metamorphosis, and if this is true we would here have an example of true myxomatous tissue in the nose. He is led to this conclusion by finding spots wherein the matrix separates into fibers, with evidence of mucous degeneration of the protoplasm of the cartilage cells in the more central portions of the growth. Guggenheim[24] reports a case which he considers benign, with photograph and description, and Moure and Pierre-Nadal[39] report a case of osteochondroma of the nasal fossæ without histology.

OSTEOMA.

A number of cases of osteoma of the nose and accessory cavities have been reported, probably occurring with greater frequency in the sinuses than in the nasal cavities proper. Marx reports three cases,[34] one originating in the anterior ethmoids, one in the frontal and one in the sphenoid. Culbert[14] also reports an osteoma involving multiple structures, including the ethmoid and frontal sinuses, while Boardman and Donovan,[7] reporting a case of osteoma of the frontal sinus, cite Sewell's paper, wherein he states that he found ninety-two cases of osteoma frontalis in the literature. Thus it may be seen that osteomata are not uncommon, but they must not be confused with bony occlusions of the posterior nares such as that reported by Rutland,[43] which are developmental defects and not new growths. Also, they must be differentiated from exostoses, frequent in the nasal cavities, especially on the

septum. Histologically they usually consist of dense lamellated bone with few Haversian canals, although they may consist of spongy bone with many vessels and abundant marrow spaces and cells.

LIPOMA.

Goldstein[23] reports the only case of lipoma of the nose or accessory cavities which the author has been able to discover in the literature. His case is described as being a fatty tumor mass in the antrum, the histology of which was reported by Thompson to be as follows: "Connective tissue stroma, holding fat cells in its meshes and enclosed in what appeared to be a fibrous capsule. Small capillaries were found in the mass, and the cells were typical of fat cells of the adult type, although some were larger than those usually found in fatty tissue."

TERATOIDS.

Of this rare condition, a true developmental phenomenon, but few cases appear in the literature, and but two with histologic description have come to the author's notice. Onodi[40] describes his case as consisting of stratified squamous epithelium, hornified, papillated and containing hair follicles and sweat and mucous glands. The stroma was formed of hyaline connective tissue, containing fibroblasts. Haenisch,[25] in 1916, gave virtually the same description of his case, except that it contained muscle, mucous glands and serous glands and adipose tissue. In addition, Gaudier[22] reports a case of cyst of the middle turbinate of dermoid contents and concludes that it was probably congenital. These tumors are not teratomata, being too simple in their construction for that term to be properly applied to them, but the first two evidently fall into the classification of nasal teratoids, the latter being, of course, a dermoid. They are probably of little more than passing interest, as their occurrence is so rare that no one individual is likely ever to encounter one in practice.

CONCLUSION.

From the foregoing it would appear that our classification of nasal growths should be revised and the revision adopted by rhinologists as a whole in order to avoid the confusion

resulting from the loose application of terms with indefinite meaning. Thus we should be enabled to differentiate nasal neoplasms according to their histogenetic and pathologic characteristics and not according to their morphology as we are now constrained to do. First, of course, we have to contend with the question, very general among pathologists, as to what constitutes a true neoplasm and whether any of the benign growths in the nose are really neoplastic when histogenetically considered, for so close a relationship exists between the products of inflammation and new growths in the nose that it is at present too difficult a task to differentiate them. It would seem, however, that for practical purposes as rhinologists a classification accepting nasal growths as they are and based on their genetic and histologic characteristics would be sufficient and certainly helpful.

And finally, although many of the conditions mentioned herein are rare, it is the opinion of the author that if more rhinologists had a slight acquaintance with the histopathology of the nose the number of reported cases would be greatly increased. It is not always sufficient to refer specimens to a clinical pathologist, for his knowledge of gross nasal pathology may be insufficient to aid him in the recognition of conditions which, while quite common in general pathologic diagnosis, might be rare and interesting specimens when considered in relation to their origin and causation.

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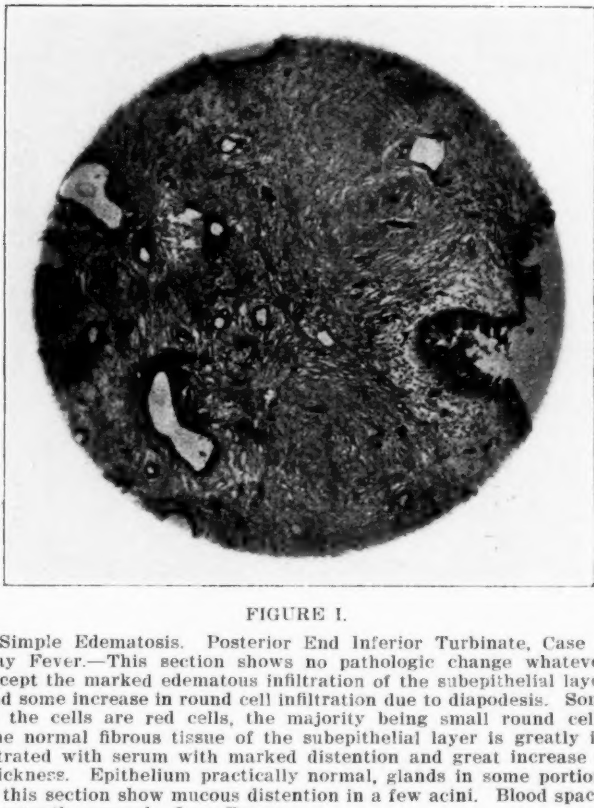

FIGURE I.

Simple Edematosis. Posterior End Inferior Turbinate, Case of Hay Fever.—This section shows no pathologic change whatever, except the marked edematous infiltration of the subepithelial layer, and some increase in round cell infiltration due to diapodesis. Some of the cells are red cells, the majority being small round cells. The normal fibrous tissue of the subepithelial layer is greatly infiltrated with serum with marked distention and great increase in thickness. Epithelium practically normal, glands in some portions of this section show mucous distention in a few acini. Blood spaces apparently normal. Low Power.

FIGURE II.

Simple Edematosis. Polypoid Middle Turbinate, Chronic Ethmoiditis.—Distention not so acute in this specimen, although it shows no apparent increase in fibrous tissue. Round Cell infiltration apparently normal. A number of dilated gland acini noted, no evidence of vascular congestion. Epithelium practically normal. Low Power.

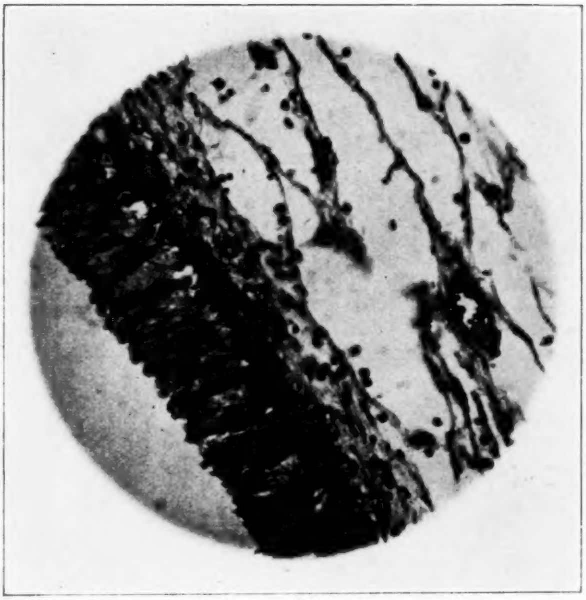

FIGURE III.

Simple Edematosis. Chronic Ethmoiditis, Lining of Cell.—Note marked edema distending fibrillar structure. Normal columnar epithelium, slight increase in round cells, small blood vessel apparently normal. High Power.

FIGURE IV.

Edematous Fibroma. Middle Turbinate.—This section shows marked fibrous hyperplasia with slight edematous infiltration. Other sections of same growth show more marked infiltration. Mucous glands show considerable dilatation in places, in others normal. Dilated glands show retained secretion and in places marked fibrosis around glands with obliteration of gland structure. Blood vascular spaces over full and at places extravasations have occurred, possibly caused by operation. Fibrosis also marked around vessels in places. Epithelium normal. Low Power.

FIGURE V.

Edematous Fibroma. Septum.—Various sections through this growth reveal marked fibrous hyperplasia with but slight edematous infiltration in most places, although same is more marked in others. Increase in usual amount of round cell infiltration and many blood vessels evident. No mucous glands are seen in this growth, probably because of its origin affording fewer such and the effect of fibrous tissue overgrowth in obliterating them. Two interesting changes are noted in this specimen; one that the surface in places shows marked folding from pressure to resemble papillary fibroma but without hyperplasia of epithelium and the other that this section shows metaplasia from normal columnar to stratified squamous epithelium. Other regions of same growth show normal stratified columnar. Low Power.

FIGURE VI.

Edematous Fibroma. Epithelial Metaplasia with Hyperplasia.—This section shows strikingly changes in normal epithelium possible in the nose. Although subepithelial structure shows nothing but typical edematous fibrillar tissue, the normal columnar epithelium is replaced by stratified squamous with marked hyperplasia until the section suggests papillary fibroma. This epithelium dips down into the subepithelial stroma but is distinctly separated from it by indefinite basement membrane. Study of the whole section shows that this is simply the epithelial covering of an edematous fibroma, enormously hypertrophied but not making up the body of the growth itself. Low Power.

FIGURE VII.

True Cyst Formation.—This section shows the formation of a mucous cyst from obstruction of the opening of mucous gland occurring from fibrous tissue formation in the substance of an edematous fibroma of the middle turbinate. Cyst contains mucous secretion and is lined with glandular epithelium, flattened from pressure of cyst contents. Note marked fibrous tissue hyperplasia around cyst and numerous smaller and one fairly large cyst in same section. Note also surface epithelium dipping down close to cyst wall from folding of growth. Very Low Power.

FIGURE VIII.

Pseudocyst of Edematous Fibroma.—This section shows formation of pseudo-cyst from accumulation of fluid in ordinary edematous polyp, so-called. The contained fluid has been coagulated and shrunken from fixing solution. Very Low Power.

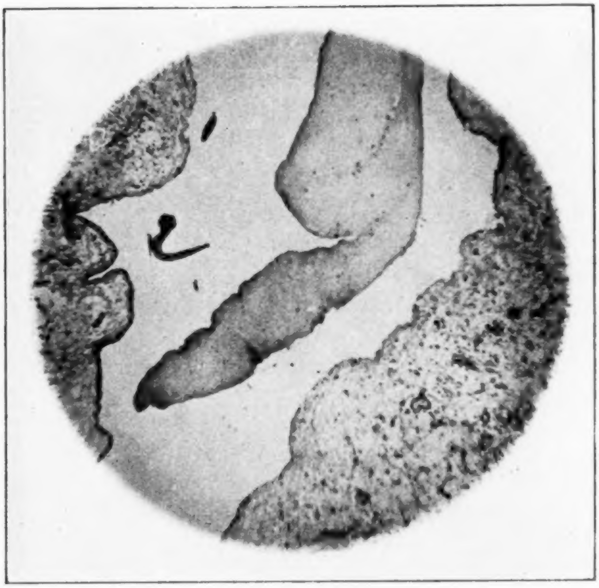

FIGURE IX.

This section taken from above slide to show endothelial lining of pseudocyst and edematous structure of the growth, with its delicate fibrous reticulum, and pseudo-cyst cavity formed in the center from accumulation of fluid in connective tissue space. Note slight cell content of fluid. Low Power.

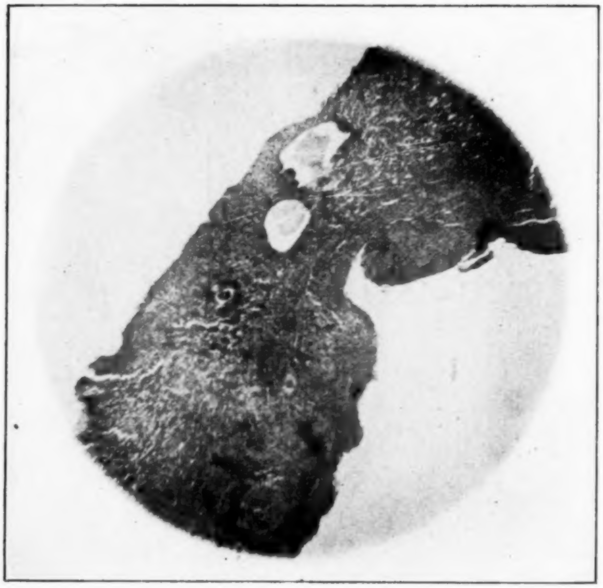

FIGURE X.

Lining Membrane from Antrum in Case of Nasopharyngeal Polypus with Cyst Formation in Antrum.—Note marked fibrous hyperplasia from old condition in antrum. Also beginning cyst formation from dilatation of mucous glands. Also marked round cell infiltration and entire absence of epithelium from pressure. Low Power.

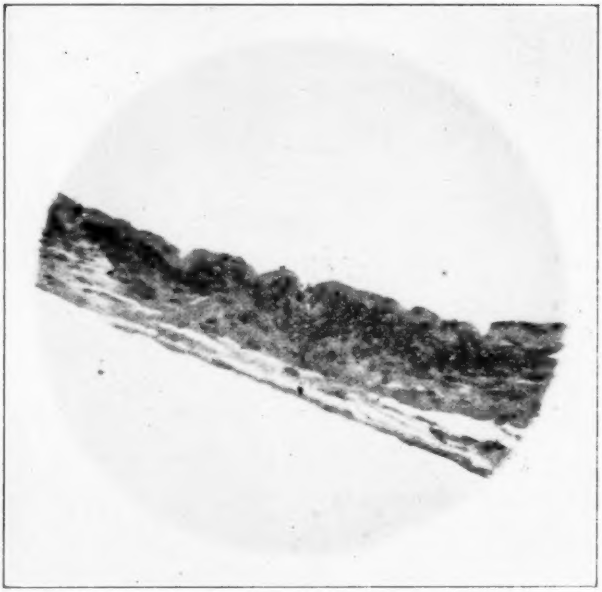

FIGURE XI.

Cyst Wall From Antral Cyst.—This section was taken from wall of antral cyst in same case as above. This cyst was probably originally a true cyst, formed by dilatation of mucous gland as shown in above section in its beginning. All trace of epithelial lining or covering has been lost from pressure, however, and cyst wall consists only of fibrous tissue. High Power.

FIGURE XII.

Papillary Fibroma, Turbinate, Squamous Celled.—Section shows marked hyperplasia stratified squamous epithelium, forming papillae with slight fibrous supportive structure, few blood vessels and no mucous glands. Definite basement membrane. Low Power.

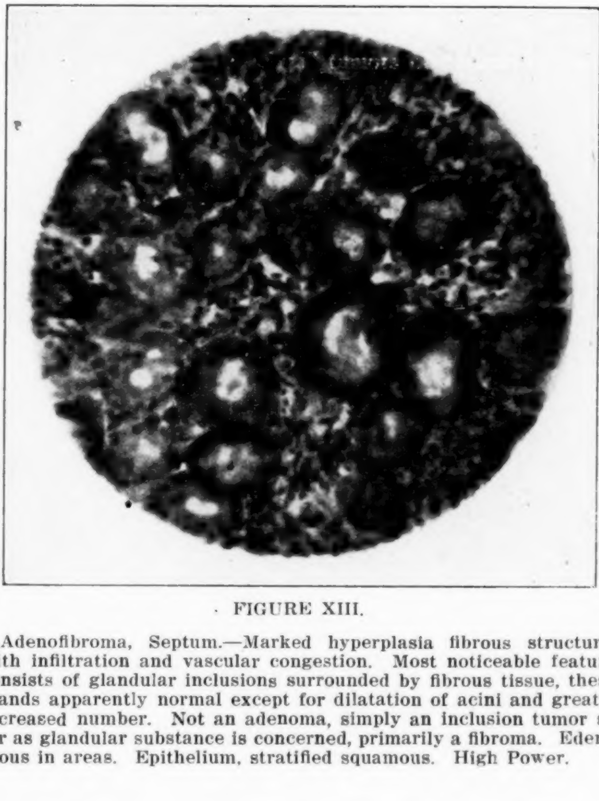

FIGURE XIII.

Adenofibroma, Septum.—Marked hyperplasia fibrous structure, with infiltration and vascular congestion. Most noticeable feature consists of glandular inclusions surrounded by fibrous tissue, these glands apparently normal except for dilatation of acini and greatly increased number. Not an adenoma, simply an inclusion tumor so far as glandular substance is concerned, primarily a fibroma. Edematous in areas. Epithelium, stratified squamous. High Power.

FIGURE XIV.

Cavernous Angiofibroma.—This section shows in other parts numerous capillaries with proliferation of their walls to form new vessels or grow in long strings of endothelial cells often several rows in depth. A comparatively thin stroma of fibrous tissue is present. This field shows most clearly the enormously dilated vessels filled with blood, with endothelial proliferation between the cavernous spaces and fibrous tissue proliferation in their walls including proliferating endothelial elements and some fibroblasts. Some hemorrhage in places. Low Power.

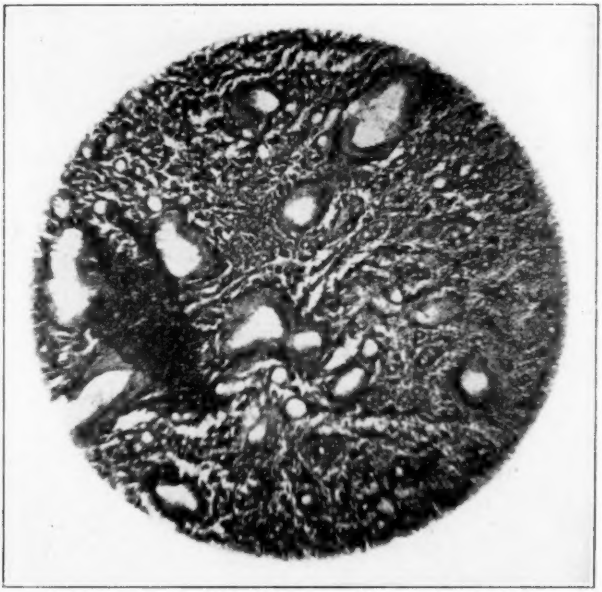

FIGURE XV.

Simple Angiofibroma.—Capillaries and arterioles surrounded and supported by fibrous tissue overgrowth. Some thickening of vessel walls, especially the larger vessels. Some of the smaller vessels show no fibrous tissue in wall whatever, being formed entirely of endothelium. No endothelial hyperplasia is noted in this section, few cellular elements and no hemorrhage. Low Power.

FIGURE XVI.

Papillary Hypertrophy Anterior End Inferior Turbinate.—This section shows marked fibrous tissue overgrowth in subepithelial layer with no edema. Fibrosis evidently old with increase in small blood vessels and apparent lessening in normal number of blood spaces with marked fibrosis around them and around larger vessels. Marked increase in round cell infiltration in subepithelial layer. Mucous glands appear to be normal in number with many dilated acini. The surface of this section shows papillated effect from fibrous tissue hyperplasia. Epithelium practically normal, stratified columnar, following configuration of surface without hyperplasia except as necessary to accommodate to increase in surface area from papillation. Low Power.

XXVII.

NASAL TUBERCULOSIS.

By W. B. Chamberlain, M. D.,

Cleveland.

That nasal tuberculosis is an unusual type of infection is undoubtedly true; that it is far more common than is generally supposed and can be looked for in the experience of almost every rhinologist is the firm belief of the writer. In 1911 he reported two cases before the annual meeting of the Ohio State Medical Association. These cases were subsequently published in the Ohio State Medical Journal and in the Laryngoscope.

During a series of years some ten or twelve cases were seen in dispensary practice, the diagnosis in each case being confirmed by microscopic examination. As accurate records in regard to these cases are not available, they are not embodied in this report. Three cases occurring in private practice, together with full data regarding them, are herewith recorded.

Description.—Confusion still exists in regard to the terms tuberculosis and lupus. Most writers regard the latter term as a relic of the days before the tubercle bacillus was recognized as an etiologic factor common to both processes. Others attribute the presence of the term to the attitude of the dermatologists in applying it to a tubercular lesion of the skin, with which a nasal involvement is often associated.

Killian, in discussing Lennhoff's paper, still insists on the use of the term lupus, inasmuch as the nasal form is always attenuated and characterized by great chronicity. Not infrequently the lupus of the face is simply an extension of a similar process in the nose. This chronic form he considers relatively common, whereas the rapid form, with extensive ulceration, is exceedingly rare—Killian having seen only one case. The vast majority of authors, however, have entirely abandoned the term lupus and speak only of nasal tuberculosis.

There is also great variability in the description of the growth. Onodi divides the process into three groups and speaks of the (1) infiltrative, (2) ulcerative, and (3) proliferative types. Freer adopts a still wider classification, common to many authors, the (1) ulcerative, (2) the solitary tumor form, (3) the diffuse swelling, and the (4) granulating—progressing superficially; while Körner describes the (1) circumscribed tumor, (2) the diffuse infiltration of the membrane, and (3) lupus. The line of distinction between the two latter forms does not seem exactly definite or clear. Possibly the least confusing and simplest classification is that of Zarniko. This classification is adopted by most authors. Zarniko speaks only of nasal tuberculosis, which he classifies into two main groups: (1) the ulcerative, occurring as an end infection in emaciated individuals and complicating a preceding process in lungs, pharynx or larynx, and (2) the tuberculoma or proliferative type, usually regarded as primary, and met with in strong, robust individuals, giving no other evidence of tuberculosis. The divisions made by others he regards merely as variations under the latter group.

Symptoms.—Individuals with nasal tuberculosis as a rule seek relief on account of nasal obstruction. Usually this is the chief, if not the only, complaint. In addition Körner mentions purulent secretion, often accompanied by slight bleeding and a tendency to crust formation. Such cases are accordingly rarely seen early, but only after the process is fairly well advanced. Practically all of my own cases have consulted me on account of nasal obstruction, though more or less secretion has usually accompanied this. Bleeding and crust formation have rarely, if ever, been mentioned. Pain is rare, except in the ulcerative form.

Appearance and Location.—The growth as a rule is situated on the anterior part of the septum and is usually unilateral. It was bilateral on one of my cases only. Out of 164 cases Mygind reports 77 per cent as occurring on the septum. Next in order are the inferior turbinal, the nasal floor and lateral nasal wall. Körner describes syphilis as a process peculiar to the rear of the nose, tuberculosis to the anterior part, the extension to the rear, or bony framework, being sécondary. One dispensary case I remember as occurring on the anterior

part of the inferior turbinal on one side only, with no involvement of the septum. The opposite side of the nose was normal.

The appearance is that of a graulating mass, mamillated and covered with more or less purulent secretion. On examination with a probe it bleeds fairly readily. The bleeding, however, is not so free as with a malignancy, nor is it as likely to persist. On pushing deeper bare bone is rarely felt. Its presence would suggest syphilis rather than tuberculosis, or possibly an intercurrent syphilis. In addition, the granulations are pale with a very definite demarcation from the surrounding tissue and no surrounding area of inflammation.

Etiology.—Most authors lay stress upon the probable infection from the finger nail in removing crusts from the nasal septum. The possibility of air borne infection should of course be kept in mind. When one considers that the nasal vestibule is the part of the respiratory tract with which infected air first comes in contact infection in this location might be expected more frequently. Factors in its prevention are probably the slight bactericidal action of the nasal secretion, together with the filtering action of the vibrissæ and the flow of the secretion outward. Infection might also occur by extension from the lower part of the respiratory tract, as well as by blood and lymph channels.

Pathology.—Tubercle bacilli are practically never found in the secretion and are often not demonstrable in the tissues. If found at all they are always rare and only discovered after prolonged search. Schultz in describing my first two cases speaks of "the number of tubercle bacilli as being surprisingly small. It required prolonged search through a large number of sections to find an occasional isolated, acid and alcohol fast bacillus. From the nature of the lesion and the numerical relationship of the bacilli, one gains the impression that the bacilli are not only not numerous but also not very virulent." Of the histologic picture Schultz says: "Of the various elements which may be present in the tuberculous process—tubercle formation with giant cells, caseation, lymphoid infiltration and proliferation of fixed tissues—infiltration with lymphocytes predominates and characterizes the lesion, whereas caseation is less marked in both cases than one usually finds in such an amount of tuberculous granulation tissue."

Zarniko's description is so concise that I give it verbatim. "The growth consists histologically of a diffuse heaping up of round cells in which single typical giant celled tubercles are scattered. At times the tubercular structure is not so evident. Then the existing giant cells of Langhans lie free in the granulation tissue. Tubercle bacilli are so rare that they frequently are not to be found, even after prolonged search. The tendency to caseation is slight. The covering consists usually of stratified pavement epithelium, which here and there sends processes into the depths of the tissue."

Differential Diagnosis.—Nasal tuberculosis is most likely to be confused with syphilis, though Onodi performed a radical operation on a case supposedly malignant, which he found subsequently to be tuberculous. Zarniko calls attention to the following differences:

1. Tuberculosis involves primarily the cartilage—syphilis the bone.

2. Fetor is rare in tuberculosis—common in syphilis.

3. In tuberculosis there is no inflammation of surrounding parts—marked inflammation of surrounding parts in syphilis.

4. In tuberculosis headache and involvement of the trigeminus rare—common in syphilis.

5. Tuberculosis is a slow process—syphilis a rapid one. In addition may be mentioned the Wassermann reaction, the administration of potassium iodid, the injection of tuberculin, as well as the histologic examination. The general examination will often prove of value as demonstrating evidences of tuberculosis or syphilis in other parts of the body. Zarniko calls attention to the fact, however, that we may find syphilis in a tubercular subject, or vice versa.

Treatment.—This is entirely surgical and consists in removal of the granulating mass with snare, punch forceps or the sharp curette. When located on the septum Körner advises the resection of the involved area by a wide incision through the septum, leaving of course a large septal perforation. Subsequently the areas are treated with lactic acid on account of its selective action, or the use of superheated air as devised by Holländer.

The prognosis is, as a rule, favorable, leading to early healing and cicatrization, though some writers speak of frequent recurrences. All of my own cases, with one exception, have yielded readily to this treatment with no recurrences. One case has been followed for a period of ten years. One case of the ulcerative type was lost sight of, so that the ultimate outcome is uncertain. This was the only case where pain was complained of. The growth has been removed with forceps and curette well into the normal mucosa, the cartilage forming the base of the area. Healing has been prompt, with no septal perforation.

REPORT OF CASES.

Case 1.—Jessie W., college junior, was first examined in October, 1914, on account of obstruction of the left nose. There was much thick secretion on both sides. High up on the cartilaginous septum on the right was a small punched out ulcer two millimeters in diameter, surrounded by an area of granulation tissue. On the left was found a somewhat larger and more superficial ulcer, with a larger amount of granulation tissue. The Wassermann was negative. There was no reaction following the administration of potassium iodid. General examination showed an old healed tuberculous process at the right apex. Two weeks after the first examination the mass on the left was removed over an area two centimeters in diameter and completely down to the cartilage. This tissue was submitted to Dr. David Marine, who pronounced it tuberculosis, tubercle bacilli being demonstrated. The lesion on the left was completely cicatrized eight weeks after operation. Eight months later there was slight recurrence on the left and a fair sized mass on the right. Both masses were removed. Four weeks later there was complete healing. This case was followed some ten months later. At that time there had been no recurrence.

Case 2.—J. M. S., physician, was first seen in February, 1921, complaining of nasal obstruction on the left. A submucous resection had been performed some years previously. Examination revealed some mucopurulent secretion, beneath which was a cauliflower, granulating mass two centimeters in

diameter. There was slight bleeding on probing, but no bone was felt. General health was excellent and no history of syphilis. Wassermann not taken. The mass was removed and submitted to Dr. Howard T. Karsner, professor of pathology in Western Reserve University. His report is as follows: Specimen consists of several small masses, some of which are covered by a stratified squamous epithelium which in some places shows elongated papillæ, which, however, do not appear to be other than proliferation due to inflammation. The tissue is made up of a connective tissue network, in some places dense and hyalin, and in other places loosely arranged. Scattered about are numerous tubercles with centrally disposed epithelioid cells surrounded by lymphocytes. In about half the instances typical Langhans giant cells are found. The density of arrangement of some of the connective tissue indicates that the process is chronic rather than acute. Diagnosis: Chronic miliary tuberculosis.

Case 3.—Mrs. A., aged 38, was examined in February, 1921, on account of left sided nasal obstruction. This was found to be due to a pale granulating mass situated along the anterior septum. There was little or no shrinking with cocain and adrenalin, and no bleeding. No bare bone was felt. This mass was removed, but unfortunately was lost. One month later a granulating mass along the anterior part of the inferior turbinal, together with a necrotic mass posteriorly, was removed. This was submitted to Dr. H. Goldblatt, resident pathologist at Lakeside Hospital, who reported the following: Histologic description—Sections of tissue from inferior turbinal. The superficial epithelium is of the stratified columnar type. These epithelial cells are very markedly swollen, and their cytoplasm is either clear or vacuolated. The epithelium varies in thickness and over a large portion of the tissue is deficient. In the subepithelial tissue there are several large and small areas of necrosis surrounded by giant cells of the foreign body and Langhan's type, by epithelioid cells and by lymphoid cells. Where there is no necrosis there is rather marked vascularity and marked diffuse infiltration by lymphoid cells, eosinophiles, plasma cells and endothelial cells. In some areas there are present large collections of epithelioid cells surrounded by giant cells. Throughout the tissue there is some diffuse

fibrosis. In the absence of a positive Wassermann, and in the absence of other signs of lues, this could undoubtedly be considered as a tuberculous process, but vascularization and fibrosis, however, make the diagnosis of tuberculosis in the absence of this information rather difficult. Special stains for tubercle bacilli will be made and an additional report sent. Diagnosis: Granuloma, probably tuberculous.

XXVIII.

THE PROBLEM OF MIDDLE EAR MECHANICS.

CHAPTER II.

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The first chapter of this paper introduced some of the facts of comparative anatomy and physiology of the middle ear complex and their relation to the inner ear. The mechanics of the mass displacement theories was discussed by taking the recent theory of Wrightson as a type. Seven leading questions were asked and answered in some little detail. It may be well to summarize the conclusions reached in Chapter I by repeating these questions and giving a brief answer to each.

I. Do the members of the ossicular chain act as an intensifying press which decreases the amount of excursion at the stapedial footplate in response to the drum membrane displacements occasioned by sound vibrations?

Reactions in the drum membrane and ossicular chain to actual plus and minus pressures in the air of the external canal and middle ear have been demonstrated. The drum membrane displacements arising from pressure fluctuations and from contraction and relaxation of the M. tensor tympani and the chain of events which appears to follow these displacements in the dead animal have been applied directly to the conditions as they obtain in the living animal and to the problem of sound transmission. This has been done without reference to the power, the frequency and the distance through which sound pulses of minimum or optimum amplitudes operate against an aperiodic apparatus. The experiments of Koehler will be considered in detail in Chapter III. The work to be done lies beyond the force applied under the conditions imposed and with the type of apparatus which must respond. The sound transmission apparatus gears down the drum mem-

brane movement, but this function is not related to sound transmission. The double lever system of the mammal makes an altered mass response in relation to sound transmission physically inconceivable.

II. Is the antagonistic action of the M. tensor tympani and the M. stapedius responsible for a balance in the ossicular chain?

Wrightson has followed the conception of the muscle function as proposed by Politzer without considering the recent work of Mangold and of Kato. If we are to assume that the ossicular chain is balanced by the antagonism of the two muscles, then these muscles must be reciprocally innervated. The contraction phase of one muscle must be accompanied by a relaxation phase in the other. It has, however, been demonstrated that the M. stapedius may contract independent of either contraction or relaxation of the M. tensor tympani. It has also been shown that the M. stapedius will hold its contracted condition on a stimulation of the M. tensor tympani. Further, in birds, the single M. tensor tympani represents a physiologic peculiarity in that we find a striped skeletal muscle without an opponent.

The writer has described the function of the passive traction system of elastic ligaments associated with the incus as positive return agents against which the contraction of the M. tensor tympani operates. The statement that the M. tensor tympani and M. stapedius are opponents cannot be accepted. These two muscles do not balance the ossicular chain in the mammals any more than does the single M. tensor tympani in birds balance the columellar apparatus.

III. Is the intratympanic pressure equal to that of the atmospheric?

Wrightson assumes that intratympanic pressure is normally equal to that of the atmosphere, and yet he furnishes a reflex mechanism which is supposed to maintain this relation. He does not consider the possibility of tubal closure increasing the pressure within the middle ear, nor does he consider the possibility of air absorption creating a negative pressure in this cavity.

IV. Is the otic capsule filled with liquid and membranes to be regarded as an inelastic container apart from the fenestra

vestibuli and fenestra cochleæ, and what relation have these two windows in preserving the balance of perilymphatic pressure which Wrightson assumes is that of the atmosphere?

The otic capsule may not be regarded as an inelastic container; the liquid and membranes may not be considered incompressible; and the two windows do not have the function of regulating the perilymphatic pressure in the manner described. The relation of atmospheric pressure to that of the perilymph is an essential feature in the Wrightson theory but it is not a fact.

V. May the membrana basilaris be considered a transformer of vertical into transverse vibrational displacements at the hair cells because of the relation of stapedial footplate and the membrana tympani secundaria, and because the route through the basilar membrane offers less resistance than the liquid displacement through the helicotrema?

This inferred functional relation rests upon two premises, neither of which can be demonstrated. First, if the membrana tympani secundaria shows no response to the contractions of the M. tensor tympani, it is logical to assume no response will occur to any mass displacements in the drum membrane brought about by sound vibrations. Second, the amount of resistance at the helicotrema is computed with the idea that there are no other pressure regulating factors save a compensation movement at the membrana tympani secundaria. Other regulating factors have been noted. The Wrightson basilar membrane response is therefore a hypothetic solution for the hypothetic mechanic assumption which in itself is not in accord with the facts.

VI. In what manner does the Wrightson piston action of the basilar membrane occur when the sound vibrations are transmitted through the temporal bone to the inner ear?

Wrightson considers the otic capsule inelastic to the forces which drive the stapedial footplate into the oval window. In spite of this he contends that the condensations and rarefactions in the petrosum as a result of bone transmitted sound vibrations may give rise to a pressure response in the perilymph which results in a thrusting of both window areas into the tympanic cavity. We are therefore confronted throughout the Wrightson theory with an involved series of elastic

and inelastic containers and a compressible and incompressible liquid membrane filler in all possible combinations. This appears essential to make the theory conform with the observed data.

VII. Wherein does the comparative anatomy show a confirmation of the Wrightson theory?

The weakness of the confirmatory evidence proposed by Keith consists in the strength which his evidence gives to the contention that the structure of any organ is dependent on its ancestral pattern. The similarity of inner ear structure in various animals in no way confirms the Wrightson theory any more than it does a dozen others. It has been mentioned that the theory does not meet the requirements in birds as well as it does in mammals. This is particularly true in those forms which have a membrana tympani secundaria which is practically extratympanic in position. The appeal to the comparative anatomy by Keith practically avoids every consideration of the mechanics of the Wrightson theory save the possibility of the basilar membrane response. Keith offers no detailed account of wherein the comparative anatomy of the middle ear region may help us to understand the obscure relations in the higher forms.

The displacement theories of hearing are based on the premise of an indirect activation of the end organ. They assume that the ear mechanism operates something like the phonograph which, as Wrightson has stated, engraves a molar displacement. The drum membrane accordingly swings in and out and transfers this movement through the ossicles to the perilymph. The shiftings in the perilymph are transferred to the end organ through displacement of the membrana basilaris or the membrana tectoria, or both, and are compensated at the round window. We have presented objections to this conception and have stated there is no evidence that the restorative forces in this aperiodic apparatus are sufficient to produce the end result. We therefore present the mechanics of the second possibility which assumes that part of the energy striking the drum membrane is transmitted as a sound pulse into the ossicular chain and is discharged as a sound pulse into the perilymph. The theory supposes that all mass movements which arise in the drum membrane, due either to excessive

sounds or to other causes, are damped out before they reach the perilymph.

The presentation of another method of sound transmission may perhaps appeal to the reader as new. As a matter of fact, however, it is so old that it has been practically forgotten. Carlisle[9] in 1805 makes the following statement: "It does not appear that any degree of motion ever subsists between the ossiculæ auditus as wholes which bears any relation to the peculiar vibrations of sound; but rather that the different motions of these bones only affect the membrana tympani and alter the degrees of contact in their articulation so as to influence the intensities of violent impulses; sounds of less impetus, not requiring such modulation, are transmitted through the conducting series by vibrations of the integrant parts of these bones, unaccompanied by muscular action. This reasoning is suggested by the columella in aves and amphibia."

Johannes Mueller,[28] in his Handbuch der Physiologie in 1840, devotes some space to careful experiments on the behavior of sound vibrations in reference to the problem of molecular transmission. Several interesting statements are made concerning the transmission of sound in terrestrial vertebrates. "Sound vibrations in air undergo a pronounced damping in being transmitted into water but pass readily into water through a tense membrane applied to its surface." "Sound vibrations in air pass readily into water if the tense membrane is applied to a short solid body which alone touches the water." "A small solid body which is applied to the water through a window in the container and is attached movably through a membraneous border transmits sound vibrations into the water more readily than other solid parts. The transmission is, however, rendered more effective if the small movable solid is attached to the central area of a tense membrane which is flanked on both sides by air." "Sound vibrations pass more readily from a membrane to a freely movable solid to water than from membrane to air to membrane into the water. Comparing the conditions with those of the tympanum, greater intensity in transmission will occur through drum membrane, ossicular chain, oval window to the liquid of

the inner ear than through the air of the tympanum through the membrane of the round window."

The fountain of knowledge in so far as it pertains specifically to the middle and inner ear behavior in relation to sound is none too limpid. It took the writer six years to precipitate any very tangible information out of a voluminous and detailed bibliography. Accordingly it may be reasonably safe to assume that any other individual of average intelligence might experience similar difficulties. The effort made in this paper is to present the facts and the viewpoint clearly. The suggestion of the simple telephone as a solution for the drum membrane and ossicular chain complex in the transmission of sound must therefore be considered somewhat in detail. The object is to describe the reactions to sound vibrations—not merely to name them. We may agree with Zimmermann when he says that the drum membrane and ossicular chain are not directly related to sound transmission. It is therefore necessary to analyze the behavior of the energy coming through the air to the drum membrane and the route by which it travels to the inner ear.

VIII. May some of the energy of the sound pulse impinging upon the drum membrane be transmitted to the liquid of the inner ear as a sound pulse and without mass displacement in the apparatus?

Let us consider our sound transmitting system in the form of the primitive wooden stethoscope—with a wide flared end applied to the patient and the smaller flared end applied to the external auditory canal of the observer. The sound vibrations arising within the patient are readily transferred to the wood and pass to the opposite end because wood is an excellent medium for sound transmission. The vibration in the wood will tend to remain within it rather than to pass into the surrounding air. It will appear at first sight that the efficiency of this form of stethoscope might be increased if the interval between the aural end and the drum membrane were filled with water. This, however, is not the case, as may be readily illustrated by the insulation which a drop of water affords to the discharge of the vibrations of a tuning fork when it intervenes between the stem and the resonator. It may also be demonstrated by filling the external auditory canal with

water and applying the stem of the vibrating fork directly to the liquid. The experiments performed by Mueller, who filled the external canal with macerated paper, may be duplicated by placing the tip of the finger in the external canal. We may apply the base of the vibrating fork to the metacarpo-phalangeal joint. The sound intensity through this source is considerably greater than if the fork is applied to the mastoid or the cartilage of the ear with the finger in place. That the finger furnishes an excellent medium for the transmission of sound vibrations may be observed in placing the tip of the finger on the resonator and by applying the fork to the knuckle. The response in the resonator is directly affected by the amount of pressure on the finger tip. In other words, the bone and cartilage transmission is excellent, but the connective tissue acts in the same manner as soft rubber. Sound transmission is rendered more efficient through compression of both connective tissue and rubber.

The tip of the obturating finger discharges the sound vibration into the air of the external canal. It also confines the pressure to this area by not allowing an escape through the external meatus. It will appear from this alone that the drum membrane affords an excellent catchment area area for sound vibrations in air. It must also be noted that a large amount of the energy of the sound vibrations entering the external canal is normally damped out through reflexion and through escape of energy toward the outside. This point will be considered in detail later.

We must therefore consider the string telephone. We will indicate the behavior of energy schematically, in a diaphragm (drum membrane) supported by a bony ring (annulus), connected through a bone-cartilagerod (sound transmission system) to a hole in the bony container (otic capsule) which includes a liquid (perilymph). It must also be remembered that the rod is insulated from its window by the interposition of elastic connective tissue. The scheme presented in the accompanying figure 2 indicates the behavior of the energy, and the entire surface of the drum membrane will necessarily be affected by the wave front striking upon it.

The sound wave (SW1) strikes upon the surface of the diaphragm (D), which is supported by a ring of bone. The

diaphragm separates the air-containing middle ear from the external auditory canal (E.A.C.). The sound wave impingeing upon the drum membrane will be reflected back and may therefore be represented in two quantities: (a) showing the back reflection and (b) indicating the quantity entering the substance of the membrane. The quantity (b) will tend to remain within the diaphragm or will pass through it as (d). The (d) quantity has a reflection back at the inner surface of the membrane owing to a decided change in medium (d1). The remainder of (d) will pass on through the air of the tympanic cavity to strike against the otic capsule (O.C.). Its course will be interrupted at every change of medium: mucous membrane (d2), connective tissue, periosteum, bone (d3), and finally periosteum (d4) before it enters the perilymph (d5). The sound vibrations entering the bone may perhaps also follow the lamellar grain because the transmission is more rapid in this direction. The amount of energy transmitted to the perilymph will be materially reduced. It is unlikely that any appreciable amount of the energy enters the perilymph through this route under normal conditions with drum membrane intact.

The quantity (c) will travel out toward the periphery, where a certain amount would be reflected back (c1), and a certain amount will enter the annulus (c2).

The quantity (e), made up not only of a part of (b) but also added to by c1 and d1, will pass to the sound transmission element. Part will reflect back at the area of the change in medium (e1), and part will follow the bone-cartilage rod to the oval window. Here it will be more or less insulated from a discharge into the petrosum, not only in the reflection back at (e2), but because the elastic tissue is a relatively poor transmitter unless it is under tension (see later). The quantity (e3) will therefore arrive at the perilymph (P1).

The quantity (SW2) indicates the behavior of the energy striking the drum membrane from the cavity of the middle ear. Apart from a difference in the behavior of the quantity d and a which have changed places, the results will be about the same. The small insert figure merely emphasizes the mechanical relation of the second opening in the otic capsule (round window) to a large mass displacement in the dia-

phragm. The round window is not necessarily associated with the transmission of sound vibrations.

It must be remembered in considering this scheme that we are regarding the transmission of the energy. It may be likened to the transmission of sound vibrations from a diaphragm along a tense iron wire to another diaphragm. Compare this figure (2) with that reproduced from Wrightson's book (Fig. 1), in Chapter I.

Disregarding the quantity (c), it will be seen that the bone-cartilage system affords a more efficient means for discharge of the energy within the diaphragm than air on either side. The force passing through as (d) not only undergoes more numerous reflections but must pass through a more efficient transmitter, bone (O.C.), into a less efficient one of the perilymph. This is naturally disregarding the various surfaces in the (d) route which may be multiplied in the mucous membrane on the diaphragm and the mucous membrane and the submucous tissues of the otic capsule in bringing the relations over the animal condition. It must also be noted that the physical character of the membrane in comparing periphery and center will be altered by the relative tension exerted. All other conditions being the same, the energy will travel in the direction of greatest tension, whether the diaphragm be pushed out by the rod or pulled in. Let us again liken the condition to the string telephone. The diaphragm tension must be greatest at its application to the transmission system. This tension is indicated in the diaphragm (D) by stippling. It will also be observed that the efficiency of the apparatus is not dependent to a great extent on the original direction of the sound wave. The same amplitude (S.W2) applied to the inner surface of the diaphragm, disregarding the dead area of the attachment of the transmission system, will give the same result as (S.W1).

This diagrammatic representation of transmitting the sound vibration answers the argument of Zimmermann[43]. Zimmermann contends that we disregard the drum membrane and ossicular chain as transmitters of sound. He does not explain the decreased hearing capacity in individuals with lacerated drum membranes.

IX. On the basis of the string telephone transmission system, in what manner are sound vibrations influenced in their passage from the air through the middle ear complex?

The suggestion of the molecular nature of sound transmission was first noted in detail by Johannes Mueller. It has recently been specifically championed by Boenninghaus[6] in his interpretation of the relations in the water-adapted mammal, the whale. This mammal keeps the regions of the external auditory canal submerged and presents a form in which the effects of mass movements in the water upon the perilymphatic space may be eliminated. The range of acuity of hearing in the whale is, after all, a doubtful quantity in spite of the well developed end organ, the large auditory nerve and the extensive crossing of the axones from the cochlear nucleus (corpus trapezoideum). The whale displays a notable departure from the usual middle ear picture in mammals in that the external auditory canal is obliterated. The small rudiment of the lumen near the drum membrane is filled with desquamated epithelium; the ossicles are heavy and united; the stapedial footplate is joined to the massive petrosum through synchondrosis; and the fenestra cochleæ is rudimentary. It may also be mentioned that the petrosum is markedly separated from the remaining head bones through spongy bone containing air cells which are continuous with the cavum tympani.

Boenninghaus contends that the entire system is directly opposed to mass movements in so far as sound transmission is concerned. He also believes that hearing may only result in terms of molecular vibration, to which the writer agrees. However, our conception of what the whale actually does hear and a conception of what sounds might arise within the water are necessarily somewhat limited. At any rate, the development of the internal auditory system may not be considered a safe criterion. However, the proposal that the condition represents one of adapted efficiency to transmit vibrations from the water to the perilymph is open to contention on the basis of the diaphragm-rod theory. In particular this is true when the inferred functional relations in the whale are carried over, as Boenninghaus has done, to the remainder of the mammals. Boenninghaus believes that the stapedial footplate is

directed through contraction of the M. stapedius, so that vibrations are discharged toward different points of impingement on the vestibular wall. This accommodation proposition is entirely beyond a physical and physiologic possibility.

The writer is in absolute agreement with Boenninghaus' interpretation that the end organ is affected only by molecular vibrations and not by mass movements. The functional adaptation placed by Boenninghaus on the whale's apparatus is, however, not in keeping with the physical requirements. Whatever a whale does hear, it probably hears through bone transmission other than the ossicles. It lacks an efficient drum membrane, and the synchondrosis of the stapes will tend to diffuse the energy transmitted into the petrosum rather than concentrate it on the perilymph. The adaptation is therefore not one which makes use of the old ossicle chain. It is rather one which eliminates the ossicle chain from the function of sound transmission. The massive development and persistence of ossicles are therefore no better criteria of a functional requirement than the development of the internal mechanism. Let us remember the preliminary paragraph in this article and the statement that "a persistence of phylogenetic structure and phylogenetic function must therefore be carefully borne in mind. Both structure and function may persist in a system without reference to its apparent office at the present time and may be entirely overlooked because the 'adaptations' appear to be complete and the present relation of structure to function too plainly obvious."

The physical requirements for sound transmission may be fulfilled with a proper catchment membrane and a stethoscope-like cartilage-bone transmitter which is insulated from the petrosum by an annular ligament so that it may transmit its energy to the perilymph. There is still another point to be explained. This is in reference to Zimmermann's work in which he deduces that the drum membrane and sound transmission system do not function for sound propagation but merely act as dampers. Zimmermann,[43] while he agrees with Boenninghaus on the molecular nature of the sound transmission, disagrees violently with the latter in practically every other detail. The interesting statement of Zimmermann that sound vibrations pass directly through the middle ear and through the

otic capsule to the perilymph is in keeping with the writer's diagrammatic representation of energy behavior (see Fig. 2d quantity). So far we are in agreement. In so far as a consideration of this as an efficient route, let alone *the* efficient route, is concerned, we are absolutely in opposition. Let us therefore look into a matter which practically all of the exponents of mass movement have completely disregarded and which Zimmermann has not considered. The general physics of both propositions is the same and must be clearly understood.

The efficiency of catchment of energy in the diaphragm will depend not only on its physical character, its shape, its tension and its thickness, but also on its position in reference to the wave front striking upon it. It should therefore be placed parallel to the wave front so that its thickness is in line with the displacing force applied to it. Let us consider this disregarded point in some little detail, because no drum membrane in nature is placed in this position. The position of the binaural hearing with the attention directed toward the source of the sound, as determined by eyes and nose, places the external auditory canals roughly at right angles to the line from the source of the sound. Further, no animal possessing an external auditory canal has a drum membrane placed at right angles to the axis of the canal. It is always found in a very oblique position. Let us assume a drum membrane placed at 45° to the axis of the canal. The efficiency of this membrane toward stopping the energy in wave front entering and traveling parallel to the long axis of the canal would be markedly reduced to what it would be if the membrane was at 90°. However, not only is this canal markedly curved and crooked but the orifice of the canal lies almost parallel to the direction of the sound wave from its source. The result of this condition is that not only a mass movement force be tremendously interfered with but necessarily a molecular one as well. This may be stated in another way: the amplitude of the original sound pulse at the external canal must very considerably exceed the amplitude of drum membrane reactions.

The laboratory of nature, particularly on the basis of natural and sexual selection in reference to sound, has been subject to great experiments. Yet we find no animal in which

the drum membrane is placed at right angles to the direction of the sound. Can one imagine a phonographic registration resulting through a horn the diameter of the diaphragm? Suppose the horn were turned about on itself like a reproducing phonograph with a small external opening, and with the singer singing across this opening and not into it. Clearly, as Boenninghaus has well stated, the phonograph and the ear mechanism are different in their reactions.

Immediately two mechanical factors suggest themselves: First, if the most efficient position of the catchment membrane is at right angles to the line of thrust, then if the membrane is placed obliquely the amount of surface offered to the thrust must be markedly increased; second, the reactions in the membrane will not only be influenced by its size and position but also by its relative size to that of the external opening in the auditory canal. It is found that in all higher forms the drum membrane is placed obliquely to the axis of the external auditory canal. Its area computed at a right angle position is also considerably greater than that of the available external opening. In other words, the megaphone of a phonograph recorder is reversed. The big end of the horn is at the diaphragm while the small end is roughly at right angles to the line of the sound source.

The phonograph represents a type of instrument in which mass movements resulting from sound vibrations are efficiently recorded. The recording phonograph operates through the mass reactions in a diaphragm which responds to intense sound vibrations. The apparatus of the middle ear, however, operates under conditions where no phonographic registration of sound vibrations will occur. The entire apparatus of the middle ear is not physically constructed for the rapid response essential to efficient mass reactions. Further, when sound vibrations of sufficient intensity are exhibited to give rise to mass responses, such responses are either inhibited or entirely "damped out."

Zimmermann's contention that the drum membrane and ossicles are not the means of transmitting sound vibration might perhaps be thought to be in harmony with the foregoing statement, but this is not the case. Zimmermann is basing his contention on the assumption that sound vibrations pass

through the drum membrane, practically as if it were non-existent, and that the sound vibrations are transmitted through the otic capsule to the perilymph. He is therefore not concerned with the efficiency or the inefficiency of the drum membrane. He merely disregards it in favor of a better and therefore more efficient route. This conception is somewhat like that proposed by Kleinschmidt.[22 23]

It appears from Zimmermann's work that although he holds the molecular propagation of sound vibrations, he misconceives the behavior of the sound pulse in reference to the physical character of the media through which it passes. Moreover, he has not considered the working of a string telephone. Inasmuch as this last point has been thoroughly considered, it is essential that we analyze the efficiency of bone transmission in the sense employed by Zimmermann.

It was shown in Fig. 2 that the (d) factor was reduced through a failure of a more rapid transmitter (drum membrane) to yield its energy readily to the less rapid transmitter (air). It was also pointed out that a reflection back occurred at every step in the change of medium dependent on its coefficient of density/elasticity. Let us eliminate the drum membrane and bring the full energy of the sound wave against the medial wall of the otic capsule. The course of the sound pulse through mucous membrane, connective tissue, periosteum, bone, finally periosteum and perilymphatic membrane will involve an innumerable series of reflections and dampings. As soon as the sound vibrations enter a rigid material like bone they may tend to remain within it through reflection back of the insulating connective tissue. They may tend, moreover, to follow the lamellar grain rather than pass across it. A great amount of energy therefore may be translated from a longitudinal to a transverse direction because sound vibrations behave in this manner in solids but not in liquids. The thickness and intrinsic structure of the petrosum may also limit the amount of energy discharge into the perilymph. The reason that individuals with stapedial fixation and normal end organ are deaf is not explained by Zimmermann. We may deduce that they are deaf because amplitudes of sufficient intensity and low pitch may not be introduced into the external canal under normal conditions to affect the perilymph through the

petrosum. This, at least, does not appear possible with the drum membrane intact.

The drum membrane, by reason of its position and structure, interferes with the sound vibrations passing through it from the external auditory canal. It does so because of the change in medium through which the energy must travel and because of the marked reflection back from its surface, and from that of the tortuous canal itself (see later).

The problem of the damping effect upon sound vibrations in their transmission to the perilymph is a complicated one. It is well to indicate that at least four phases must be considered: first, the effect of the shape and position of the external auditory canal; second, the damping effect of the drum membrane to sound vibrations passing through it into the cavity of the middle ear; third, the effect of drum tension on the sound transmission apparatus; and fourth, the damping effect of muscle contraction.

The photographic registration of sound pulses has been markedly improved within the past few years. In particular, the behavior of the sound pulse in reference to its passage through various tubes and membranes, by Foley of Indiana University,[14] are well worth a close study by otologists. The following four photographs, presented through the courtesy of Professor Foley, shows the silhouette of a sound pulse which may be likened to serial photographs of a distending soap bubble. In the later, a limiting membrane is formed by a film of soap and water. In these photographs a limiting zone is created by the refraction of light caused by condensation and rarefaction in the air. The limiting zone of condensation (dark) and rarefaction (light) is therefore a criterion of the enormous amplitude in this short wave length produced by an electric spark. The sound pulse in the first three photographs, taken at about 1/1,000,000th sec., shows the behavior of the distending pulse in its passage through four kinds of schematic tubes: A straight cylinder (a); a megaphone with convex walls, small opening in and large opening out (b); a megaphone with the large opening toward the sound source (c); and a crooked tube (d). The reader will remember the dark band is in advance of the light band and indicates the direction of transmission. (Figs. 3, 4 and 5.)

In following the progress of the pulse through the straight tube (a) it will be noted that but little reflection occurs. There is but little decrease in the width of the band and practically no "bowing" in the wave front. Compare this with the megaphone (b). Again we find little evidence of reflection but a marked "bowing" of the wave front and a consequent dilution of amplitude due to the "side-slip" or diffraction. This "bowing" of the wave front may be readily seen when the position of the band at the edge of the megaphone is compared with that of the sound pulse in the straight tube. Compare this with the megaphone (c) and observe the enormous reflection back from the curved sides. Practically all the energy is reversed except that in the small area traveling on through the end of the megaphone. This small area compares favorably both in position and intensity with that of the corresponding point in the straight tube.

It is interesting to note the behavior of the sound pulse traveling through the crooked tube. Here an enormous dilution in the original amplitude takes place through reflection of the energy at the convex sides of the tube. The position of the wave at the opening corresponds to that in the straight tube (i. e., the velocity of the sound is dependent on the medium through which it is traveling) in spite of the irregular course. The energy of the sound pulse as indicated in the width of the band has been materially damped out.

These photographs demonstrate the damping effect of a crooked tube very convincingly. In likening the behavior to the conditions in the external auditory canal, it must be remembered that in the photograph we are dealing with a single pulse of great amplitude and extremely short wave length in which the wave front enters the opening directly. Whereas, in the case of the external ear, we are dealing with much smaller amplitudes, much greater wave lengths and a wave front which enters the canal almost entirely through diffraction. It is not unlikely that a diagrammatic canal may be constructed and placed in the form and position of the human external ear and external auditory canal. If the same amplitudes were applied as in these figures, the amount of energy arriving at the lateral opening indicated in Fig. 5 in the crooked tube would be so greatly reduced it might even fail

to register by this method. And in any event, were a delicate membrane placed across this opening no photographic registration of a sound pulse passing through the membrane might be observed (see Fig. 6). The diffraction of the sound vibrations into the external auditory canal is undoubtedly supplemented through reflection from the external ear, as Kato's experiments seem to indicate (see later).

We may carry over this information to the report of Burnett's[8] experiments which were undertaken in Helmholtz's laboratory. The sound vibrations in this work are carried over from the source by means of a rubber tube 1 m. long. The aural end of this tube was connected with a glass tube fixed in the external auditory canal. "The position of the glass tube in the external auditory meatus has a great influence on the experiments. When the tube is directed downwards and forwards, the experiments were almost invariably successful, but in any other position they may be partially or entirely unsatisfactory. For in the former position the sound waves strike directly against the membrana tympani and hammer, whereas in any other position they are forced against the sides of the auditory canal and are deflected or destroyed before they reach their destination."

It must be remarked in this type of experiment the external auditory canal was closed to the outside and the escape of back reflected energy through the external meatus was limited. The amplitudes of the sound vibrations conveyed in this manner are probably beyond normal intensities. The writer experienced no difficulty in hearing a low pitch fork attached to the end of a meter long rubber tube under similar conditions, even when the tube itself was twisted. It also appears mass drum membrane reactions arise only when the route to the membrane was rendered straight so that a direct impingement of intense sound vibrations might take place.

Nagel and Samajloff[30] made similar observations by means of the clever arrangement of connecting the middle ear region in such a way that it was so interposed in the feed to a sensitive flame. "It was found necessary to maintain an open pathway through which the sound vibrations might pass to the drum membrane, and the tube, tied in the external auditory canal, was placed under tension so that the canal could be

rendered straight." "We applied a vibrating fork upon a cranial area corresponding to the head of the malleus and observed pronounced reactions in the flame. This was more pronounced when the external auditory canal was closed. This may be considered new evidence of the incorrectness of the Harless notion that the interpretation of the Weber and Wheatstone experiments (that a tuning fork is heard more loudly through the teeth when the external auditory canal is closed) might be regarded as a deception or error in judgment. The experiment further demonstrated that vibrations in the bone are carried over to the air of the external auditory canal and from this to the drum membrane."

Nagel and Samajloff also observed that similar reactions took place in the sensitive flame to vocal and bone transmitted sounds. It is not unlikely that the Harless interpretation is not a correct one. The transmission of vibrations through the finger in the external auditory canal with the fork applied to the knuckle illustrates this in a simple manner. It is also probable that bone transmitted sound vibrations normally pass through the ossicular chain. On the other hand, the experiments of Nagel and Samajloff were performed under abnormal conditions. They probably were dealing with abnormal intensities in a dead animal, and the explosion waves which arise in speaking or whispering into a tube were not ruled out. The detailed article of Harless on "Hearing" is worthy of careful consideration by any student of this subject. Harless was a prominent exponent of the theory of molecular transmission and even diagrammed the "centering" of the energy upon the footplate of the stapes.

Dennert[11] is also an exponent of the molecular transmission of sound vibrations through the ossicular chain. He makes an interesting series of objections to the experimental evidences on the mass movement reactions. We take the liberty of quoting from his work because his article has not received merited attention:

"An attempt has been made to confirm the theory of mass movements in the sound transmission apparatus by means of ear models and through the findings in normal and pathologic specimens. These experiments are indeed germain because they indicate the interrelation of the entire intratympanic ap-

paratus including the labyrinth contents in toto. A movement of the apparatus, including the contents of the labyrinth, has been established with the manometer as a result of gross mechanical causes, such as the experiment of E. Weber, in which pressure was exerted upon the apparatus through the finger; those of von Helmholtz, in displacements of the manubrium mallei in a model constructed by him; in the registration of plus and minus pressures; and under the influence of very powerful sound vibrations. But these observations do not demonstrate that similar mass movements of the sound transmission apparatus, including the labyrinth contents, actually occur under normal conditions of hearing, because under normal circumstances we are dealing with extremely minute power factors. The observations on the phonograph have been regarded as an important substantiation of the mass movement theory, and it may not be denied that these observations are apparently in accord with the ideas of mass movements in the sound transmission as applied to the ear. But here also, as has already been mentioned in regard to the experimental investigations on models and preparations much greater forces are required, and the demonstration is one which shows a tremendous superiority of the ear over the phonograph in relation to sound vibrations of small intensity. According to Morak, whispered numbers may be correctly understood at a distance of 89 m., but they are not registered by the phonograph even when spoken directly into the megaphone. The vast superiority of the ear over the phonograph may still better be demonstrated in the quantitative method of testing hearing devised by myself in which, with a low pitch fork, a certain number of vibrations are permitted to impinge upon the drum membrane in a certain unit of time. While one of my forks with a vibrational duration of 30 sec. may be picked up as intermittent in passing it by the external canal three or four times a second, practically throughout its entire duration, the phonograph, in spite of its diaphragm and its lever mechanism, fails to record this same fork when vibrated continuously deep in the megaphone. We may therefore regard the observations on the phonograph as analogous to the above mentioned observations on models and specimens and as gross mechanical processes of sound transmission, while the molecular trans-

mission to the ear is a more ideal, and much more delicately refined process connected with minute forces in the sound vibrations." This may also be regarded as an answer to the Wrightson contention that the inner ear must operate under similar conditions to those under which the phonograph registers the molar movements of its diaphragm.

The damping effect of the drum membrane on the sound vibrations impinging upon it and being transmitted through it into the air of the middle ear also deserves mention. According to Zimmermann, the membrane offers little or no resistance. Professor Foley obligingly made a photograph of the reflection back of a sound pulse projected against a delicate collodion membrane probably not one-fifth the thickness of a human ear drum. (Fig. 6.) This membrane was supported by a wire holder which appears as a heavy dark band. The amount of reflection back is enormous, and if the reproduction of the figure is successful only a delicate line will complete the circle on the far side of the wire holder. This may be taken as evidence of two things: First, the actual damping effect of a membrane, and second, the very small amount of energy which is required to stimulate the inner ear through the sound transmission system. If one can overhear the conversation in an adjoining room through a closed door it is probably safe to assume that not 1 per cent of the energy in the first room passes into the second through the door and walls.

The damping effect of the drum membrane will therefore mean on the basis of the diaphragm-rod theory that only a very limited amount of the energy transmitted through the external canal will pass into the drum membrane. This will necessarily limit the amount of transmission of sound vibrations to the perilymph because of the small and inefficient catchment area.

It has been demonstrated that the position and character of the external auditory canal has a decided damping effect on sound vibrations. These vibrations must enter through diffraction or a sideslip in the wave front of practically 90°. It was also shown that a large amount of the original energy entering the external canal is reflected away from the drum membrane area. It has been indicated that only a small

amount of the energy striking upon the drum membrane enters that membrane. A large part is reflected back into the external auditory canal. It was also pointed out that the drum membrane yields but little of the energy it receives to the air of the tympanum. We are now prepared to consider the relation of the tension of the membrane in reference to the problems of sound transmission into the ossicular chain. The relation of drum tension to the amount of back reflection must be considered and the influence of drum tension upon the transmission of energy into the ossicular chain also taken into account.

It has been shown by Mangold and Eckstein[28] that "even if a true damping of sound vibrations may not be disputed and has been established during the voluntary contraction of the M. tensor tympani in E. (an individual), we must, however, emphasize that neither of us received the impression that an irritating sound stimulus is damped and rendered less irritating through the tension of the M. tensor tympani as determined by the noise of its own contraction. Even strong tensor contractions, which lead to distinctly recognizable tensor noises as well as to demonstrable pressure variations in the external auditory canal, show themselves powerless to damp the disagreeable sensation. The assumed protective reflex against strong and high pitch sound stimuli is even more problematic in individuals in which the contraction of the tensor in its assumed reflex function is not subjectively recognizable and, until this time, could scarcely be classed as objectively demonstrable."

The increase in drum tension, according to this statement, does not produce a pronounced damping effect. This must be interpreted according to the diaphragm-rod theory. The string telephone operates through a tense membrane and with its most tense area at the attachment of the transmitting element. If the medial displacement of the malleus increases the tension in the connective tissue of its periosteal attachment to the drum membrane, it will also increase the efficiency in transmission of sound vibrations from drum membrane to the malleus. Any damping effect therefore which may come about must be referred to the less efficient catchment through a more tense drum or to the subtraction of energy through the tensor

tendon itself. Connective tissue under tension is a better transmitter than nontense connective tissue.

The point that the efficiency in the sound transmission apparatus depends in part on the positive pull of the malleus against the drum membrane is not merely a speculation. It lends itself well to the interpretation of the deafness caused by a hypertense drum membrane. Hypertension is usually due to the medial displacement of the drum membrane through air absorption or through adhesions. The connective tissue between the drum membrane and malleus is relaxed in these conditions and sound transmission from drum membrane to malleus impaired. It appears that the M. tensor tympani through its contraction will tend to damp out mass movements through the increased "weighting" of the drum membrane itself.

Both the Burnett and the Nagel and Samajloff experiments appeal to the writer as insufficient in the determination of the optimum, and in particular the minimum energies. Both series of experiments appear to show that mass oscillations of the drum membrane may arise under circumstances of unusual intensity in sound vibrations. One would not test the efficiency of a dictaphone by removing the mouthpiece some distance from the lips and whispering softly in its general direction. The two experiments noted in the previous paragraphs demonstrate the "damping out" effect of the external auditory canal and the drum membrane, even when the sound vibration is conveyed directly to the external canal. The damping effect will be more pronounced when the sound pulse undergoes a lateral dilution in its "side slip" at an angle into the external auditory opening. We must also bear in mind the surprisingly small amount of additional damping which even large masses of cerumen occasion. This will be discussed in Chapter III.

Kato has considered the problem of the "damping out" of the sound vibrations in their transmission through the ossicular chain, and the reader is referred to his article for details. "While it has been more or less generally accepted, in spite of the evidence to the contrary presented by Bockendahl, that the intrinsic muscles respond to sound vibrations with lightning-like contractions, comprehensive and repeated experiments have demonstrated that both of the intrinsic muscles respond

with prolonged contractions as long as the sound may operate." "Opposed to this, however, are the findings that actual medial displacement of the ossicular chain does not occur as a result of the reflex contraction of the M. tensor tympani ,because the M. stapedius always responds and fixes the stapes. The M. tensor tympani reacts to stronger stimuli while the M. stapedius responds to weaker intensities; the contraction of the former is therefore always accompanied by reaction in the latter. However, no actual medial displacement of the stapes ever occurs, as has already been pointed out. This would only take place if the M. tensor tympani contracts independently, under which circumstances (with the M. stapedius out of commission) a medial displacement of the stapes may be observed in the reactions of the membrana tympani secundaria. Inasmuch as muscle contractions arising from sound vibrations never occasion an increase in the perilymphatic pressure, the 'damping out' effect suggested by Zimmermann (in terms of increase in the tension of the membrana basilaris) may not be considered."

"In conclusion, some mention must be made of the manner in which the intrinsic muscles perform their function of 'damping out' the intensity of sound vibrations. There are two possibilities: either the fixation of the stapes and the more marked drum tension results in a decrease in the amplitude of the vibrating masses, or a diminution of the intensity with which the sound vibrations are transmitted to the labyrinth is brought about through these changes. Naturally it is possible to conceive that both mechanisms may operate."

Kato's experiments on the reflex reactions to sound vibrations in the rabbit and the cat are extremely instructive. They give a possible test for acuity and range in hearing in various forms which is much more satisfactory than any hitherto known. The cat responds reflexly to pitches of 200 to 300 vibrations per sec., while the rabbit reacts to 128 and occasionally to 64. The fact that the M. stapedius contracts to 50,000 vibrations per second, which is entirely beyond the range of hearing in the human being, indicates a molecular transmission rather than a mass response. It is impossible to conceive a mass response in sound transmission apparatus to a frequency of 50,000.

The rabbit reacts to the pitch A3 at a distance of 60 m., but if the external cartilage is removed this distance must be cut down to 29 m. Removal of the drum membrane reduces the distance still farther to 8 m.; while reflex contractions are, however, again provoked with the pitch A4 at a distance of 5 cm., with malleus and incus gone and even with the stapes removed, reaction in the muscle may be observed if the source is applied immediately to the external canal. This evidence is very important in considering the efficiency of the damping effect of the external canal, and also the efficiency of the membrane and ossicular chain route for the transmission of sound vibrations as opposed to the direct route through the medial tympanic wall or round window as suggested by Zimmermann and others.

The normal cat under certain test conditions becomes reflexly deaf in about five hours, while rabbit under similar circumstances is deaf in from five to six minutes. The time is markedly reduced if one of the muscles is out of commission, and still more markedly reduced if both muscles are cut or thrown out of function by means of profound narcosis. It has also been shown in various mammals and birds that cochlear degenerations occur earlier as a result of the exposure to prolonged high pitch vibrations in animals with the intrinsic muscles incapacitated than in those with muscles intact. Objections may be made to these experiments in that the animal is subjected to abnormal conditions of sound intensity. The extent of the lesions in both animals and birds indicates that at least two factors operate in producing the results. First, a fatigue and possible degeneration of a small cochlear area as a result of overstimulation; and, second, the spreading and relatively indefinite area of degeneration shows that mass movements in the perilymph are also probably responsible for the greater part of the resulting lesion. The relation of the problem of experimental cochlear degeneration and its bearing upon occupational deafness may not be taken up at this time.

It is, however, permissible to indicate that further work is demanded on the problem of the damping out of sound vibrations. Attention has already been called to the effect of the position of the external auditory canal and to the "side slip" of the sound wave front entering it. Investigations also ap-

pear to show that a great amount of the original energy entering the canal and registering in the form of drum membrane responses is "damped out" through reflection from the tortuous walls of the canal itself. Foley's sound pulse photographs indicate that a very large amount of the original energy striking upon a thin celloidin membrane is reflected back toward the source. Finally, the writer has emphasized the oblique position and the physical character of the drum membrane as objections to a mass response as a result of normal or optimum amplitudes of sound vibrations. It does not appear likely that the muscle tension or the stretch of the elastic ligaments can subtract any very material amount of the energy passing through the transmission apparatus. This appears to conform with the inability, voluntary or otherwise, to actually damp-down or damp-out sound vibrations. The damping response must be correlated, however, with the experiments of Kato, where he finds a stapedial contraction to extremely high pitches (50,000). He also obtained reactions to sounds of reasonable pitch at a distance of 60 m., which is probably beyond the range of the explosion or concussion waves arising from the sound source.

There can be little doubt that the rabbit hears the sound indicated in Kato's experiment at a considerably greater distance than 60 m. The fact that the response was not elicited beyond this distance with the external ear intact or at more than 29 m. with the external ear removed will show that sufficient energy is being applied to cause mass response of some kind and contraction of the muscle to "damp out" these movements of the stapes as a whole. This is to the writer a very important point because it indicates that the M. stapedius is definitely opposed to mass movements in the sound transmission system. The amount of molecular transmission is already limited through the size of the catchment area itself and by the factors already noted. It appears unlikely under normal conditions that sufficient molecular response through the drum membrane and ossicular chain could be produced to effect an actual degeneration of the cochlear apparatus. However, the excessive amplitudes could, and undoubtedly would, provoke mass responses which the intrinsic muscles might eliminate as long as they are functional. This is equiv-

alent to saying that the cochlear degenerations arise at least for the most part from mass movements in the sound transmission apparatus due to excessive amplitudes. As long as the muscles are physiologically capable of cutting down these mass movements, the reflex deafness does not come about. It is not fair to assume that the rabbit has a keener sense of hearing than the cat and therefore becomes deaf in five minutes under conditions that produce reflex deafness in the cat in five hours. The cat may be able to damp out vibrations more readily. It may also flatten out its external ear and offer a certain amount of additional damping out to the diffraction of sound vibrations into the external canal. This is apparently what takes place, because the cat not only flattens the ears against the head but also presents its tail toward the sound source.

The deafness which comes about through lateral displacement of the drum membrane, either due to a plus pressure in the middle ear or relaxation of the M. tensor tympani, or both, is probably not due to a disengagement of the incus from the stapes, which, as Bezold has noted, breaks the contiguity in the ossicular chain. It has been mentioned that the passive traction system of incudal ligaments opposes the contraction of the M. tensor tympani. It therefore contributes to a lateral drum membrane displacement on relaxation of that muscle. The lateral displacement of the long process of the incus from the stapes will readily come about because the ligamentum annulare is a very strong stapedial attachment when compared with the delicate elastic capsular ligament at the incudostapedial articulation. This disengagement may be observed in the guinea pig, where the malleus and incus are united by bony ankylosis and where the lateral displacement of the malleus necessarily carries the incus with it.

If Secchi's observations[36] are correct, then the middle ear is normally under a slight plus pressure. A tonus contraction of the M. tensor tympani might be assumed to maintain a contiguity at the incudostapedial articulation. The problem is not to be regarded as definitely settled. The suggestion may, however, be made that drum membrane tension is, after all, only an incidental result and is not the purpose of the contraction of the M. tensor tympani. This will at least con-

form with the relations as they are observed in birds where the M. tensor tympani function does not appear to be definitely related to the drum membrane tension.

Kato's experimental evidence on reflex contraction of the intrinsic muscles and the reactions which may be interpreted in terms of acuity in hearing demonstrate that the drum membrane and sound transmission system are the efficient means of transmitting sound vibrations. This is in direct opposition to the contentions of Zimmermann, Kleinschmidt,[27] Secchi,[35] Kretschmann,[25] and others, that the transmission takes place through the air of the tympanum, either through the medial wall of the tympanum or through the fenestra cochleæ. The reader is referred to the excellent reviews of Treitel,[35] of Frütiger[17] and of Lucæ.[26]

The writer feels justified in presenting the diaphragm-rod theory of sound transmission and declaring that the mass movements ordinarily described lie beyond the physiologic maximum. In other words, the entire apparatus from drum membrane to hair cells is set in for molecular vibration. The moment mass movements of the drum membrane arise they are damped out in so far as this is possible. The sound transmission system therefore represents a most efficient area of bone transmission to the inner ear, and in particular is this true in small amplitudes (intensities) and long wave lengths (low pitch).

The registration of vibrations in the auditory cells may therefore be effected by the sound pulse passing on them or through them. Neither membrana basilaris nor membrana tectoria can effect the mechanical stimulus essential to a reaction. The fact that the auditory cells are hair cells and the fact that they are definitely related to a membrana tectoria may be due to the ancestral type of pressure organ or displacement organ from which they are derived. The membrana tectoria may perhaps offer inhibition to mass movements. It may be a sort of stabilizer. This X quantity, which occasions a reaction in auditory cells to vibrational frequency and intensity, is not particularly different from the behavior of the retinal cells. Is it really essential to our conception of the behavior of the light in the various media of the eye that the workings of the rods and cones be established? Perhaps

then we have arrived at a point where our limitations confront us. We may assure a sensitization of cells to picking up frequencies and their amplitudes. We may harmonize this behavior with the extensive researches in cochlear degenerations as a result of prolonged auditory irritation. But we may also at this time remember the words of the immortal Goethe: "Eben wo Begriffe fehlen da stellt ein Wort zur rechten Zeit sich ein."

It appears therefore that the drum membrane and entire sound transmission system is set in for appreciation of sound vibrations of small amplitudes. When amplitudes ot sufficient intensity are produced, the tiny mass vibrations of the drum membrane are absorbed and dissipated in the hinge system of the sound transmission apparatus and further inhibited by the contraction of the intrinsic ear musculature. The very minimum which the displacement theorists grant may therefore represent a maximum of hearing or even beyond the capacity of appreciation in terms of intensity of hearing.

The writer appreciates this last statement is a decided shock to anyone who has followed the mass displacement theories in any one of the various forms. Perhaps the first thought which comes to mind is this: if a balanced drum, a straight rod and an insulated opening in the otic capsule to the perilymph fulfill the requirements for sound transmission from air to end organ, why do we find a more elaborate mechanism with muscle attachments and elastic ligaments? Why is it essentialy that a fenestra cochleæ with its limiting elastic membrana tympani secundaria be found in practically all forms of terrestrial vertebrates? These questions, proposed by Beyer, may be quite successfully answered. If the explanation does not hold on the basis of comparative anatomy and physiology, then the diaphragm-rod theory cannot be employed as a working basis. If this be accomplished, the problem stands open for the tests of repeated experiment before the theory may be accepted. The essential differences between all displacement theories and that of molecular transmission consists in this—not only must the inner ear be physiologically guarded against any factors of displacement arising from sound vibrations, but all displacement factors arising in the middle ear must also be ruled out in so far as this is possible.

X. What is the function of the middle ear complex?

Keith remarks in relation to the Wrightson theory that we must seek an explanation in the comparative anatomy and physiology for matters which are not clearly defined in the human being. Objections were made to the confirmatory evidence proposed by Keith because the close agreement in the general structural pattern of the inner ear merely shows that the structure of a given part must bear some definite relation to the ancestry of the organ. This is neither a discovery of Keith's in reference to the inner ear nor does it substantiate the Wrightson hypothesis more than it does any one other of a dozen theories. It was also pointed out that the mechanical relations in the bird are less readily associated with the Wrightson hypothesis than they are in the mammal. This also obtains for any other displacement theory. The very similarity in structure will make any theory of hearing which includes the facts obtain for mammal and bird alike. In other words, if we solve the problem for one form we solve it for all forms within certain limits which are dependent on individual variations.

Curiously enough, the entire Wrightson hypothesis rests on a middle ear behavior; the mass reactions in the drum membrane; the "intensifying press" function of the ossicular chain; the muscle balance; the displacement of the stapedial footplate in relation to the membrana tympani secundaria. Yet Keith furnishes no evidence wherein the comparative anatomy such as that of another acutely sensitive form, the bird, furnishes a confirmation to the correctness of these fundamental conceptions. Perhaps therefore a study of the comparative anatomy and physiology of the frog, the bird and the mammal may furnish a clue to the complex middle ear system. It must be remembered that the middle ear region has a far more interesting phylogeny than that of the inner ear. It was stated in the beginning of this article that a consideration of the reptiles would be eliminated. This family presents an extremely variable picture which must, of course, not be disregarded. The reader is entitled to first hand information in so far as this is possible. We have not investigated the reptilian ear. Three forms have been selected—the frog, the bird and the mammal, because they represent three distinct

possibilities in middle ear mechanics. The information on the frog has been largely derived through courtesy of Terence Ahearn, S. J.,[1] who is at present writing his doctor's thesis on the problem, undertaken in this laboratory. The reader is referred to Ahearn's article when it appears for a detailed description. The work on the bird and the mammal are a result of personal investigation. The report on the elastic ligaments of the cat (dog may also be included) are presented for the first time.

A. The middle ear mechanics in the frog. (Fig. 7.)

The drum membrane in the frog is relatively thick (d. m.) and is supported by an annulus (an.) of cartilage. The sound transmission system is roughly in the form of a twisted U, with one extremity of the U markedly thickened and the connection between the two arms is swollen. The cartilage portion (lateral) displays two definitely weakened areas at which bending occurs upon light pressures. Following the apparatus from the otic capsule (o. c.), the small descending process displays a hinge at (a) and then swells to be attached to the medial drum membrane surface. This attachment area runs upward, loses its connection to the drum membrane and forms a small process which bends sharply on itself at hinge (b) to continue medialward in an enlarged club shaped mass —the columella proper—which occupies the upper area of the fenestra vestibuli. It will be noted that the surface of this process is in relation to the perilymph (pl.) and is insulated by elastic ligaments from direct contiguity with the otic capsule at its upper surface. Below it is mortised into another cartilage element the operculum (o). It is joined to the operculum by elastic ligaments. The operculum is confluent with the otic capsule at another cartilage hinge (c) so that all movements of the sound transmission system are accompanied by accompanying movements in the operculum. The outward displacement of the cartilage is resisted by a stout elastic ligament (not indicated), while the inward displacement is resisted by the M. opercularis, the action of which is indicated by the arrow (m).

The inner ear system is represented as a hole in the otic capsule filled with perilymph (pl.) and containing a ductus cochlearis (stippled). The capsule has three chief openings—

the fenestra vestibuli filled by columella, operculum and elastic ligaments. The second opening is the foramen perilymph sup. (F.P.S.), where the perilymph liquid is separated from that of the spatium subdurale (S.S.) by a thin membrane. The third opening or foramen perilymph, inf. (F.P.I.), is closed in by an elastic membrane, the membrana tympani secundaria (M.T.S.), which faces the mouth cavity but is separated from the mucous membrane by a lymph sac (L.S.) and by muscle fibers (not indicated). It will be seen that the tuba auditiva (t.a.) is a short wide open duct leading from the mouth to the cavity of the middle ear.

When the frog swallows the air, the middle ear cavity is placed under a plus pressure with a resulting lateral drum membrane excursion indicated by the arrow +. This force, because of hinge "a," is translated at "b" into a reverse motion indicated by the arrow. The motion of the drum membrane outward tends to thrust the sound transmitting system into the perilymphatic space so that increases in pressure would escape along the lines indicated by the fine arrow—through the cochlear duct or around the cochlear duct (helicotrema) toward the foramen superior and foramen inferior. Minimum motions are probably compensated for in the vascular supply. However, larger movements are prevented by the contraction of the M. opercularis. This muscle is therefore opposed to both displacement factors in the columella proper. It is also interesting to see that if a medial displacement factor is applied to the drum membrane (arrow x), the area at hinge 'b' will become applied to the drum membrane and the resulting motion in the columella will be the same as before.

The mechanism in the middle ear in the frog is a response to an adjustibility in the sound transmission system which arises because of the lateral drum membrane motion. This motion is dependent on respiration and allows a maximum excursion at the drum membrane area with a minimum response at the perilymphatic end. The active phase opposed is the plus pressure due to air injection. It may also be stated that according to the diaphragm-rod theory an inefficiency in the apparatus may be seen in the direct connection of the sound transmission system to the otic capsule, and in the thickness in the membrana tympani. The articulation of the columella

with the opercular cartilage merely indicates a large adjustment area toward preventing pressure fluctuations in the perilymphatic system. It is also necessary to bear in mind that the helicotrema in the frog is an extremely wide opening offering none of the objections of resistance to displacement mentioned by Wrightson for the mammal and bird.

While the frog does hear certain things, its sound transmission system and drum membrane do not appear particularly well adapted. Nor does the development of the inner ear suggest a refined sense of hearing.

B. The mechanics of the bird's ear. (Fig. 8.)

The schematic diagram of the bird's inner ear shows similarity to that of the frog excepting that the membrana tympani secundaria (M.T.S.) (fenestra cochleæ) has rotated to face the middle ear. It must be remembered in some birds (the goose, etc.), the fenestra cochleæ is separated from the cavum tympani by the vena jugularis. The membrana tympani (D.M) in birds is thin and markedly convexed outward into the external auditory canal; the two middle ear cavities are confluent, and the two tubæ audivitæ (t.a.) open through a common tubal duct (t.d.) into the mouth. This small opening is normally closed. The sound transmission system consists of a cartilage tripod which occupies the posterior drum area. The M. tensor tympani attaches to one element of this tripod. This muscle is extratympanic and the line of its pull is indicated by the arrow M. The muscle is resisted by elastic ligaments, notably the columellar-squamosal ligament (c.l.) of Platner. The cartilage apparatus joins the bony columella just medial to the attachment of this ligament. The bony columella (C) is attached to the margins of the fenestra vestibuli by elastic ligaments, and the membrana tympani secundaria is also elastic in structure (M.T.S.). The otic capsule is now well separated from the cavum subdurale, and a hole is left in it to represent schematically the aqueductus cochleæ and aqueductus vestibuli (aq.). It will also be noted that the membrana vestibularis (Reissner's membrane) is indicated in a heavy vascular tegmentum vasculosum (T.V.).

When the tubæ auditivæ open freely to the mouth, the air pressure on each side of the drum membrane is probably equal. Displacement forces arise with closed tuba, either through air

absorption or through positive pressure at the external auditory canal. The latter tends to thrust the columella inward and beget increased pressure in the perilymphatic spaces. This increased pressure will be absorbed by the blood vessels (note the position of the tegmentum vasculosum), or may escape through the aqueductus cochleæ et vestibuli or at the membrana tympani secundaria. The contraction of the M. tensor tympani directly opposes this and tends to hold off the inward drum membrane displacements from increasing the pressure in the perilymph (pl.). Negative pressures in the external canal are also resisted by the elastic ligaments and compensated by the great mobility in the drum membrane. There are also a series of cartilage hinges in the sound transmission system which allow a more pronounced reaction in the tripod cartilage than in the bony columella proper (see writer's article, American Journal of Morphology).

The mechanism in the bird's ear is one set in for variations in drum membrane position. The compensation system is found in an adjustibility of the sound transmission apparatus and is of direct physiologic importance. The muscle tensor tympani does not figure conspicuously in terms of drum tension, although this necessarily results. Rather the M. tensor tympanum in birds combats active displacement factors which tend to produce excesses of perilymphatic pressure. The cochlear area in birds is poorly placed to be acted upon by sound vibrations direct through the otic capsule. The range of adjustibility of blood vessels in the perilymph system is even better developed than in the mammals. It may also be mentioned that the columellar footplate does not lie at the end of the cochlear duct but faces its middle area.

C. The mammal. (Fig. 9.)

The schematic diagram presents the relations in the human being, and some reservations must be made in carrying over this scheme to the cat, the guinea pig and in particular the mouse. The two middle ear complexes are completely separated. A new cartilaginous tuba auditiva is shown as closed with the function of the M. tensor veli palatini (M. dilator tubæ) to open it indicated by the arrow M. d.t. The drum membrane is drawn in and attached to the malleus. The position of the incus and stapes is indicated. The pull of the

M. tensor tympani is represented by the arrow at M. t., while that of the M. stapedius is shown by the arrow M. s. The inner ear is about the same as that pictured for the bird and frog, which will indicate the purely schematic presentation. The heavy vascular tegmentum vasculosum in the bird is replaced by the delicate nonvascular membrana vestibularis.

When the tuba auditiva is opened, the M. tensor tympani appears to relax, allowing lateral displacement of the drum membrane. This lateral displacement of the drum membrane may be due to several factors: first, the intrinsic elastic structure of the drum membrane, which will tend to decrease the umbo-depth slightly; second, the incudal elastic ligaments and character of the incudomalleolar articulation may contribute to a positive push factor outward of the malleus; third, the manner of tubal closure may create a positive pressure within the middle ear region as Secchi has suggested; and lastly, a minus pressure in the external auditory canal (barometric) will tend to draw the drum membrane outward. It appears that the drum membrane at rest is not placed in a position of physiologic efficiency and tendency for a disengagement at the incudostapedial articulation must be counteracted by muscle pull. This mechanical requirement in the mammal does not obtain in any other vertebrate. The diagram presents a possible interrelation between the M. tensor tympani and the M. tensor veli palatini. The arrows indicate the former muscle is set in against any factors which will make for a lateral drum membrane displacement. The medial displacement of the ossicular chain tends under unusual conditions of pressure to force the stapes toward the perilymph and beget increases in perilymphatic pressure. This is opposed through contraction of the M. stapedius. The distribution of the possible excess pressure in the perilymph, apart from that taken care of in the minute veins, is shown in the arrows within the inner ear.

We must grant, on the basis of Kato's experiments, that reflex contractions of the M. tensor tympani do not give rise to fluctuations at the membrana tympani secundaria. It must also be granted that the M. stapedius absolutely opposes minute displacements of the stapedial footplate. The vascular regulator mechanism compensates for what would constitute

mass movements in the sound transmission system in reference to any motion created by responses of the drum membrane to sound vibrations of usual intensities. The compensation area in the membrana tympani secundaria will therefore not be called for in relation to either sound vibrations or to reflex contractions of the M. tensor tympani. It will only be effective in the larger drum membrane fluctuations which in the mammal are necessarily limited. It has been mentioned that the relatively larger fenestra cochleæ in birds is suggestive of a compensation area for perilymphatic adjustments to mass movements in the sound transmission system. These movements are not a result of sound vibrations but are due to barometric fluctuations and to air absorption.

The double displacement factor in mammals therefore calls for two muscles. A new muscle, M. tensor tympani, is related to factors which tend to produce a lateral displacement of the drum membrane and probably maintains a contiguity in the sound transmission system at the incudostapedial articulation. The second muscle is similar in function to the single M. tensor tympani in birds or the M. opercularis in frogs. The M. stapedius affords protection against increases in pressure through the application of a movable skeletal element to the perilymphatic space. Both of these muscles tend to inhabit mass movements in the sound transmission system.

Throughout the entire comparative anatomy of the middle ear region three prominent features appear: first, a veriability in the position of the drum membrane not dependent on hearing but on displacement factors arising within the middle ear; second, an adjustibility in the sound transmission system gearing down the mass drum membrane movements in relation to the fenestra vestibuli. These are not related to sound vibrations in their propagation to the perilymph but merely decrease the range of mass motion. Third, protection is afforded to the delicate end organ through muscle contraction against increases in pressure brought about through drum membrane displacements and in inhibition to the mass responses propagated from the drum membrane toward the perilymph. This appears to harmonize more completely with the comparative anatomy and physiology of the middle ear region than any suggestion thus far published. The work on the

intrinsic muscles of the mammal by Kato and the investigations on the human M. tensor tympani by Mangold are certain to stimulate researches along these lines. It is also important that the tubal mechanics and the function of the M. tensor veli palatini be included.

It is only fair to state that the contractions of the M. stapedius to the sound vibrations, as noted in the experiments of Kato, are not readily interpreted. We must assume that the M. stapedius is always in a state of tonus contraction. When a sound is superposed upon the sound picture of the environment to the point that it begins to occasion a mass reaction or a tremble in the stapes, an additional muscle contraction is manifested. This statement means that the stapes, because it is extremely light and because the energy is discharged from the footplate into a medium of shorter wave lengths, may have sufficient restoration forces connected with it to undergo mass reactions. The effect of the M. stapedius in its contraction, as confirmed on experimental animals, would be to act as a damper.

The comparative anatomy and physiology affords certain evidence which distinctly favors the diaphragm-rod conception of the sound transmission. It may be well to reserve judgment on whether this, that or the other theory is the correct one. It is our contention, however, that the older view of a molecular transmission has certain things in its favor. It should at least be included in our discussions of the problem. In particular, the diaphragm-rod theory lends itself well toward the solution of several features in deafness which have been considered as hopeless. Perhaps they have been considered hopeless because the mechanics of sound transmission have been looked upon as quite well established by Helmholtz.

We may take the liberty, in closing this chapter, to refer again to the opening paragraphs in this article, where we call attention to the fact that the "functional adaptation" and "structural adaptation," after all, are merely terms which indicate that we have approached the region where cause and effect may not easily be separated. It may come to pass that what we have been considering as the active stimulating factor in sound analysis—the membrana tectoria, membrana basilaris and the delicately refined histologic structure of the

end organ may, after all, be nothing more than an earmark of the ancestral pressure or displacement organ from which it was developed. The resultant microscopic picture must be followed with this point in view rather than believing it will necessarily explain a selective function in the auditory cells. Let us remember that whatever the method is by which these cells operate, even under the lowest tones, the ear is quicker in its response than the eye. The discrimination of a suppressed vibration up to a frequency in high pitches which exceed that of touch (130 per sec.), implies that marked after-images such as found in the retina do not occur. This is an important point to bear in mind, because a sympathetic vibration does not omit vibrations when the vibrations are eliminated from the activating source. The inner ear therefore appears to operate under no conceivable conditions of inertia, which indicates the activating agent as a sound pulse and not a mass response in the sound transmission system.

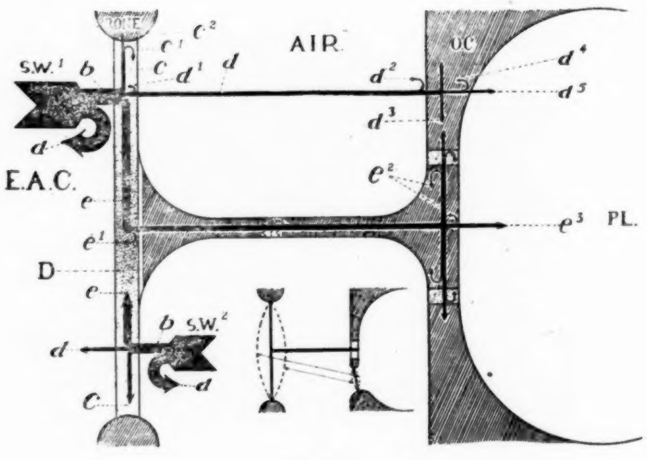
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Fig. 2.

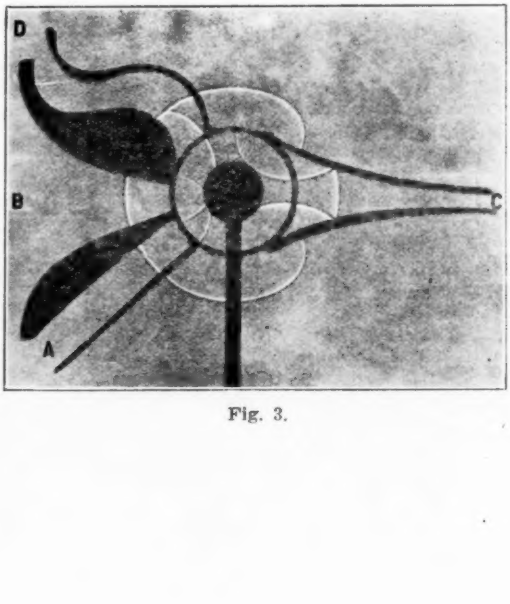

Fig. 3.

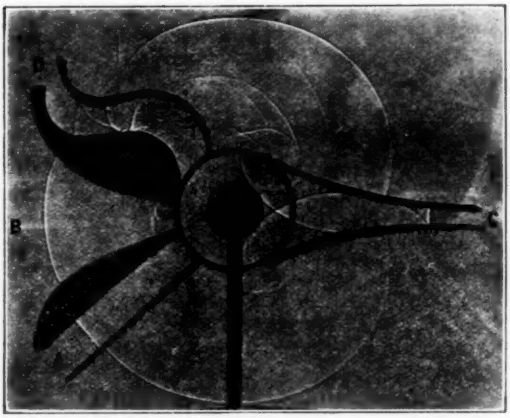

Fig. 4.

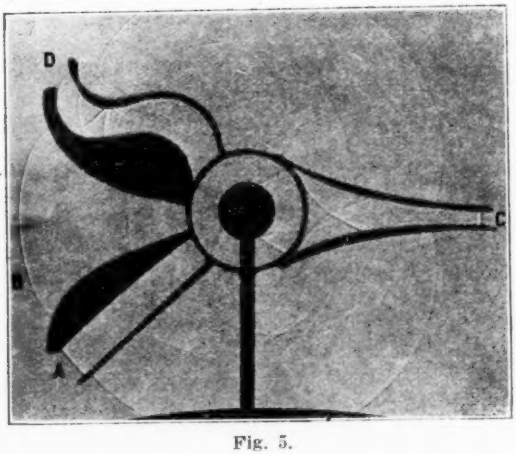

Fig. 5.

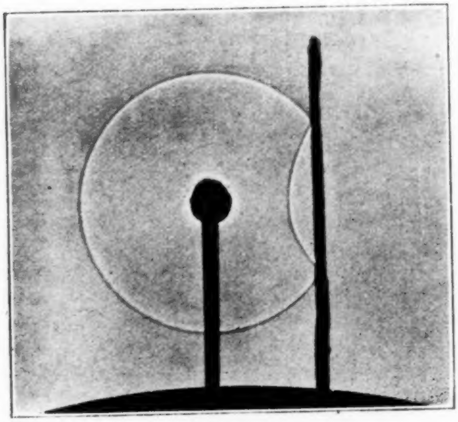

Fig. 6.

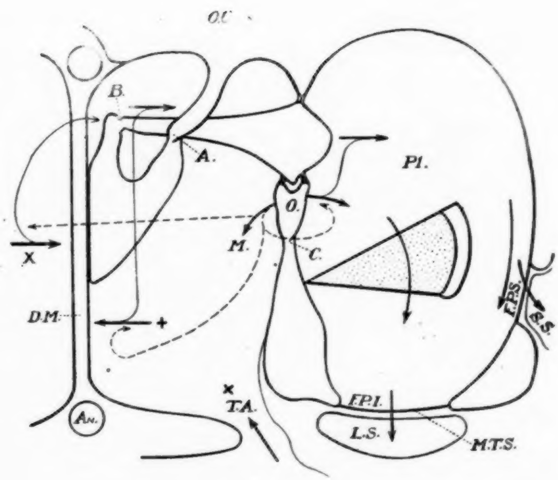

Fig. 7.

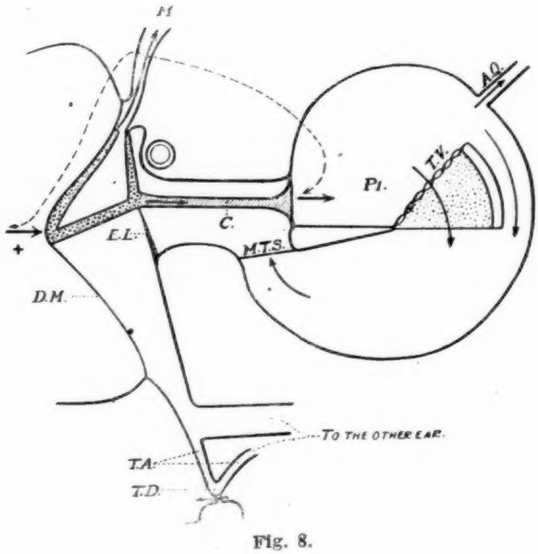

Fig. 8.

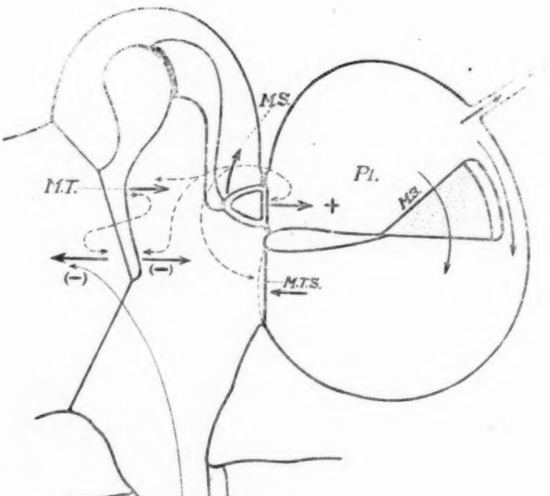

Fig. 9.

XXIX.

MISCONCEPTIONS REGARDING THE IMPORT OF RETRACTION OF THE DRUM MEMBRANE.

By Edmund Prince Fowler, M. D.,

New York City.

Elemental physiology of the ear teaches that normally the drum membrane is made convex peripherally and concave or funnel shaped centrally, by its attachment to the more mesially situated manubrium, and that the air pressures upon its two sides are approximately equal owing to free communication with the surrounding air, constant by way of the external meatus on the one side, and periodic by way of the eustachian tube upon the other.

The drum membrane is then normally retracted, its central fibres are tensified and curved from the plane of the annulus toward the manubrium, and markedly toward the umbo.

Some confusion or carelessness exists as to the terminology of abnormal drum tensions, hyper and hypotension being used erroneously to denote respectively lack of or increased mobility of the drum. As a matter of fact, hypertension denotes immobility only in the sense of an increase in strain upon the drum; whereas lack of mobility may or may not accompany such a strain. Likewise hypotension means a loss of tone or lowered strain upon the drum fibres so that increased mobility will coincide with such loss of tone only if there are not limiting adhesions, etc.

We use the term retraction to indicate an abnormal sinking in of the tympanic membrane, and as this may be due to a relatively lessened air pressure upon its inner side, it has become an almost universal habit to think of the condition as diagnostic of negative middle ear air pressure, and of the relief experienced from inflations as due to the restoration of more equal pressures upon the two sides of the drumhead.

However, when we examine retracted drums, it is amazing to find that in but comparatively few instances can any negative air pressure in the tympanum be demonstrated. Even

in marked cases, with great foreshortenings of the manubrium and prominence of the processus brevis and posterior fold, there may be no vacuum within the middle ear.*

How, then, are we to interpret retraction of the drum? I believe we may better understand the matter if we will give it a little thought along the lines I am about to indicate

The walls of the eustachian tube, being soft and practically in contact for a considerable distance, are easily approximated more firmly by inflammatory or other swellings in the mucosa or submucous connective tissues. When such occlusions of of the tube endure for some time, we are led to believe that there ensues sufficient air absorption from the middle ear and its connecting spaces to appreciably retract the drum. This inward negative pressure tensification may cause diminished hearing, often tinnitis, and at times vertiginous sensations. It also sucks together the walls of the tube (in like manner to the inspiratory effect upon the gill valve of a gas mask) and thus makes more permanent and firm the tubal closure.

If instead of or beside tissue swelling there is present an exudate in the tube, this fluid will not only tend to close the tube as such, but on account of its viscosity will cause the tubal walls to adhere, or as the direction of the cilia motion is pharynxwise and all transit through the tube can be demonstrated to be more easy in this direction (and for this reason) there will be instituted another cause for exhaustion of the tympanic air, namely, a suction brought about by the downward movement of the fluid. Depending upon the position of the head, gravity may aid or hinder this movement. I have been able to demonstrate only during deglutition and automatic movement of fluid from the middle ear into the tube.

Any lessening of pressure in the tympanum will cause the external atmospheric pressure to predominate, and the drum with its movable attachments will be forced inward. Two main factors, then, may operate to cause retraction of the drum after occlusion of the tube: First, absorption of middle

*The method for determining air pressures behind the drum was described in detail last year before the American Laryngological, Rhinological and Otological Society and the Otologic Section of the A. M. A.

ear air; second, movement of the fluid contents of the tube toward the pharynx, mainly by cilia motion, and exhaustion of the middle ear air by such hydraulic suction.

If the negative pressure is of sufficient strength, a fluid exudate is expected to fill the tympanum to a greater or less degree.

After failing to find any relative data in the modern or ancient literature, an attempt was made to determine the exact amount of negative pressure requisite to cause an exudate from mucous membranes. Small cupping glasses applied to the buccal mucous membrane gave no definite findings; in fact, different persons appear to be affected in varying degrees by like pressures. The Schneiderian membrane also gives various results under similar negative pressure in different persons. In hypertrophic rhinitis, an exudate is obtained after a short period of suction; in atrophic rhinitis but little effect can be produced even with strong suction (over 40 or 50 mm. Hg.) applied for long periods of time (one-half to one hour). This, of course, would be logically expected, and I believe similar lesions within the middle ear and tube may account for the ready, or tardy, or non-occurrence of serous or mucoserous exudates accompanying retraction of the drum membrane. In like manner, hypertrophic and atrophic otitis media must influence the ready, tardy, or nonoccurrence of air absorption.

Small negative pressures, then, do not cause an exudate into the middle ear; but from the observation of many cases I feel certain that only small negative pressures (5 to 20 mm. Hg. at most) accompany otitis, and that if there be caused marked retraction of the drum this cannot be due to their strength, but rather to the prolonged or oft-remittent presence of negative pressure, first weakening and then allowing of marked recession of the drum.

Positive external meatal or negative middle ear pressures up to 20 mm. Hg. depress the drum membrane but little compared to pathological retraction.

I have examined scores of cases with apparently a complete tubal closure, wherein no negative air pressure within the middle ear could be demonstrated by any of the methods I have described for checking this factor. There can be but

one explanation for such a failure, namely, no appreciable air absorption is occurring in these cases. No other deduction seems tenable. Such cases also frequently show little retraction of the drum. Here again there is present no negative air pressure. It is obvious that when the tube is functioning normally, no negative pressure will long endure within the middle ear spaces, and no air absorption operate to retract the drum; but with open tubes marked permanent retractions of the drum are common.

There is some doubt as to just what constitutes a patent tube. I would say that a physiologically patent tube is one which will permit automatic ventilation of the middle ear, especially during swallowing, whether the air in the tympanum is of normal pressure or of lessened pressure due to prior deflation from any cause.

Another finding is that after firmly plugging the external meatus with various cereates, etc., no absorption of air from the external meatus may be demonstrable, perhaps even after days of waiting.

By showing how slow may be the air absorption from the body spaces, it is only necessary to remind you that it takes often 8 to 12 days to absorb 1,000 to 2,000 cc. from the peritoneal cavity, after inflations incident to X-ray diagnosis.

We may thus account for the failure of meatal plugs to effectively hold or pull the drum outward through air absorption. Has any one ever examined a drum after subjection to the supposed suction from meatal plugs and found it other than in its previous retracted position? I think not. This failure occurs even when the tube is patent. Again, no appreciable air absorption has occurred from the tissues. The external meatus epithelium may be considered to approximate roughly an atrophic state of the mucous membrane of the middle ear. In such states it is reasonable to doubt the ready absorption of air.

Some one will say: Well, you will admit that the drum cannot be retracted without something holding it back. Yes, but, in a way, it may be. How, then?

For the same reason that a bowl is hollow, a section of pneumatic tire concave, a soft hat crinkly. There is insufficient tension at the periphery to maintain the fabric as a flat

surface, and the redundant material must take a position to one side or the other of the peripheral attachments. Why, then, is the concavity of the drum usually outward, if there is no difference between the air pressures upon its two sides? Because: First, during the establishment of the retraction, portions of the drum become set in this position and so remain, unless forced outward by inflation or external meatal suction. In fact, slight increases in middle ear pressure may fail to cause local retracted portions of the drum membrane to bulge outward, and strong inflations to maintain bulging but for a few minutes, far too short a time to account for their collapse by air absorption on their mesial side; second, the tube, not functioning physiologically, there is a tendency to maintain in the middle ear a stationary air pressure. This tendency is often observed, not as an active force in increasing retraction but simply as a preventive of restoration to the centered position of a weakened drum. If there were present a measurable negative pressure, constant stretching of the drum and a constant increase in the retraction would occur; but that such need not be so is evident from the many cases which apparently remain for long periods in status quo. Third, adhesions or fibrous bands between any part of the middle ear and the retracted portion. These may be weak or elastic enough to admit of quite free movement of the membrane, and yet sufficient to cause its return to the retracted position upon removal of the replacing force. They may be difficult to demonstrate.

Of other causes for retraction, I will not speak at length at this time but, needless to say, of the utmost importance are adhesive processes, displacement of the ossicles, shortening of the tensor tendon, subluxations, stretching of the ligaments, etc. All these have, I believe, more to do with permanent retraction of the drum membrane than has air absorption.

CONCLUSIONS.

Hypertension is not synonymous with immobility of the drum membrane. Hypotension is not synonymous with increased mobility.

Air absorption may be the primary cause of retraction of the drum membrane. It is never over a few millimeters

(under 20) of negative pressure, greater in hypertrophic or congested states: practically nil in atrophic states, and in the latter seldom if ever the primary cause of exudate into the middle ear. It cannot operate with patent tubes or with perforations. It often is absent with closed tubes. It is usually unmeasurable except in active stages of tubal or middle ear congestion. The lesions accompanying retraction of the drum are of more importance from a clinical standpoint than is the negative pressure.

Permanent retraction of the drum is seldom if ever due to the pressure of negative or diminished air pressure within the middle ear. This factor as an accompaniment or cause of drum retraction has been and still is greatly exaggerated.

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XXX.

ACUTE INFECTIONS INTO THE SEBACEOUS GLANDS AND HAIR FOLLICLES OF THE NASAL VESTIBULE.*

By Lee M. Hurd, M. D.,

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The common and usually trivial boil in the nose may suddenly terminate fatally by extension of the infection through the venous channels to the cavernous sinus. This is not realized by the laity and is not, I fear, any too well recognized by many physicians. When only two text books on rhinology mention furuncles and do not hint, even, that there might be grave complications, and also, when only a few text books of general surgery mention the subject, it is no wonder that this condition is considered so lightly. It is a striking fact brought out by the literature that most of the cases are only recognized when they are well advanced and hopeless, usually by the consultant who reports the case.

If anything is to be done to cut down this mortality it will be by early recognition that the cavernous sinus is threatened and by very prompt measures taken to present it.

Etiology.—Predisposing and exciting causes are similar to those of furuncles elsewhere on the skin, with the added bad habits of picking nose and pulling out the vibrissae. The staphylococcus is the common organism but it may be streptococcus.

Symptoms.—Pain, tenderness, redness and circumscribed swelling in the vestibule sometimes extending through to the skin of the external nose. Later softening at centre with rupture occurs and discharge of pus and slough "core" followed by prompt healing.

This trivial discomfort may suddenly become extremely serious by extension to the cavernous sinus, due either to

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*Read before the American Academy of Ophthalmology and Otolarygology, 26th Annual Meeting, Philadelphia, Pa., October 17, 1921.

attempts at opening, usually with a dirty needle at home, or, by squeezing, rupturing nature's barrier, driving the infection into the less resistant subcutaneous tissue, or picking the head off the furuncle with a dirty finger nail, or, by too free incision, opening up new avenues outside nature's barrier. In other cases it seems to travel through the venous channels by reason of lack of resistance to infection.

Diagnosis.—A furuncle, or collection of them, should offer no difficulty in diagnosis; in fact, the patient usually has correctly diagnosed his red, swollen, painful nose as a boil and has usually operated with a needle and squeezed it, seeking medical aid because it is getting worse under his treatment.

Complications.—There may be sloughing of the inferior lateral cartilages.

The cavernous sinus becomes infected more frequently than is generally thought and it is one's duty to forestall this calamity, if possible, for the mortality of cavernous sinus thrombosis is practically 100 per cent.

It behooves us to manage the case so that the infection will not reach the sinus, if it can be avoided.

According to the literature, one feature of nearly all the reports is that the cases were not seen until too late and diagnosis made still later. Also, that the vestibular furunculosis is not well classified, cases being in with other case reports. As to when and how the primary focus was treated is not clear enough to draw any constructive deductions, whether the cavernous sinus thrombosis came from either neglect or mismanagement, or was so fulminating that nothing could be done.

With a vestibular infection a tentative diagnosis of cavernous sinus thrombosis should be made on the appearance of any of the following eye or orbital signs which should be considered very suspicious: Swelling of the lids, chemosis, pupillary changes, deep seated headache, beginning ptosis and exophthalmos, lacrimation, congested retinal veins, symptoms of sepsis. Later, when ophthalmoplegia, pronounced chemosis, edema of the lids and exophthalmos plus cerebral and septic symptoms develop, the case is hopeless and it usually is only a matter of a few hours before the opposite eye

indicates that both cavernous sinuses are thrombosed and dissolution is near.

Treatment.—Though most of the vestibular furuncles are trivial affairs, it should ever be remembered that, from poor resistance of the patient or bad management, a seemingly trivial affair may explode with fatal results.

When fortunate to be seen early, my procedure is to use wet dressings inside and out and, as soon as there is evidence of pointing, to make a small incision at this place and never to go outside of nature's barrier, and by applying suction to draw out the infective material, fill the cavity with bismuth paste between treatments, and never to squeeze the swelling which might cause rupture into the subcuticular tissue where the chances of tissue resistance are much lower. This treatment seems to be much better than extensive incision and expression which is so often mentioned in the literature of the cases which later developed cavernous sinus thrombosis.

I have treated a number of cases in which the local involvement was severe, yet with a small incision entirely within the barrier and with suction, they have recovered without complication except sloughing of inferior lateral cartilage in three cases.

As this infection extends along the anterior facial, angular and superior ophthalmic veins to the cavernous sinus above and perhaps by the deep facial vein and pterygoid plexus below, it seems rational to ligate the anterior facial vein near the inner canthus, as Bullock[1] reports in one successful case of upper lip infection, and also to ligate the anterior facial above the deep facial vein at the anterior border of the masseter muscle as soon as the infective area is increasing instead of diminishing or, if there is the slightest orbital or ocular symptom of cavernous involvement.

As lateral sinus thrombosis sometimes recovers, similarly, why may not the cavernous sinus overcome a slight infection if further supply is stopped? At least facial vein ligation is a simple procedure and may save the patient's life.

As surgical attack of the cavernous sinus for anterior infections has up to the present failed to cure ([3 4 5 6 7 8]) it has in a few instances even failed to open the sinus, but further technical experience and earlier diagnosis will probably pro-

duce better results from the use of one of the different methods, in what is now a hopeless situation.

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XXXI.

"VARIATIONS OF INTRACRANIAL SYMPTOMS FROM FRONTAL SINUSITIS."*

By Louis Levy, M. D., F. A. C. S.,

Memphis, Tenn.

In preparing this paper I have tried in the following pages to bring out the various intracranial symptoms that are presented during or following attacks of frontal sinusitis.

Realizing from experience that in no two patients are these symptoms the same, it can readily be understood why no set rules can be applied in making a diagnosis of extradural or intradural complications.

It is natural that the malignant tendency of sinusitis may manifest itself by its extension through the cerebral wall of the frontal sinus and result in the meningeal symptom of frontal lobe abscess. It is therefore well, when these symptoms appear, to be on guard for the possibilities of cerebral complications.

In reviewing the literature regarding the intracranial complications following frontal sinusitis, it is surprising that such a small amount of attention has been given to the subject. This may be due to the fact that abscesses in the frontal lobes cause no early symptoms and paralysis takes place only when the involvement becomes generalized.

In 1914, Boenninghaus reported eighty-seven abscesses of the frontal lobe, caused by frontal sinusitis. In 1919, Gerber made a careful review of the literature and reported in all two hundred and forty cases of intracranial complications dependent upon diseases of all the accessory sinuses, of which sixty-five were brain abscesses. As Freudenthal remarks, "Many cases have occurred following operations which were never reported or either not recognized antemortem."

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*Candidate's thesis which was accepted by American Laryngological, Rhinological and Otological Society, 1922.

In reporting these cases it is not with the thought of presenting something rare, but to emphasize the many and varied symptoms to which attention has often been attracted by similar reports throughout the literature.

Case 1.—On October 7th, 1920, S. P., age 16, consulted me complaining of marked headache and swelling of left upper eyelid. He gave the following history: Cold past two weeks with severe leftsided headache past week, for the past three days left upper eyelid gradually swelling until the present and eye now closed. Marked quantity of pus blown from left side of nose. Upon examination, the patient presented the following: Left eye closed with marked oedema of left upper eyelid and conjunctiva, also some puffing of the lower lid. Eye movements, pupillary reactions, and fundi were normal. Upon palpation, marked tenderness over left frontal region. Marked hypertrophy of both middle turbinates with pus flowing from beneath left middle. Transillumination showed small right frontal sinus clear, left side, large and cloudy, antra clear. X-ray pictures confirm diagnosis of left frontal sinusitis and also show left anterior ethmoids involved. Advised patient be taken to hospital immediately for nasal operation, but parents objected. On morning of October 8th, was called by family stating that boy was having convulsions and was unconscious. Upon insisting, he was taken to the hospital at once. Temperature 101, pulse 96, blood examination gave a leucocytosis of 20,000 with 79 per cent polynuclear cells. Lumbar puncture produced a clear fluid under slight increase of tension and examination showed cell count 12, globulin normal, glucose plus 3. As patient had been given a quantity of morphin to control convulsions, and was in a stupor, under a local anesthetic, removed left middle turbinate, clearing out ethmoid cells and making large opening into frontal sinus. Sphenoid opening seen clearly and no pus from same. Patient put to bed, no packing in nose. On October 9th, patient better but claims he cannot see and upon testing out vision with fingers, found he was able to see nothing but light and shadows moving before eye. Ophthalmoscopic examination of fundi, at this time, showed slight venous distention, otherwise normal. Slight tenderness in left temporal region, temperature normal. October 10th, feeling well,

headache gone, and now able to see. October 11th, blood examination gave a leucocytosis of 19,000 with 90 per cent polynuclear cells, temperature 100, pulse 88. Patient drowsy when aroused, marked irritability. Morning of October 12th, nurse noticed that patient could not raise right leg, right arm, when gripping object, seemed weaker, however, able to use same. Tender place over left temporal region now fluctuating and was opened under ether. Found pus had broken through frontal sinus at external angle, also perforating the inner table, perforation being about the size of a dime. Dura appeared normal. Wound packed. October 13th, doing fine and now able to move leg. October 14th, blood examination gave a leucocytosis of 19,000 with 82 per cent polynuclear cells. October 15th, during the night had recurrence of convulsions, the first since primary operation. Reopened wound and found a small pocket of pus, as wound was healing too fast. October 16th, temperature now normal, patient doing splendidly, having complete control of both legs. Urine normal. A laboratory report from examination of the pus, as well as culture made, showed a pure culture staphylococcus aureus. This patient had been seen previously to this attack on May 28th, 1920, at which time his vision, fundi, and nose were normal. Patient last seen on November 9th, having completely regained his health, the operative scar barely visible.

Case 2.—On November 13th, 1920, Mrs. B., age 62, consulted me on account of a swelling over the right frontal region. Patient states that for the past year this swelling comes and goes and at times gives her no trouble. Examination reveals frontal sinus abscess, which has perforated outer table. X-ray picture confirms diagnosis. Advised radical operation, on which patient decided to wait. November 22nd, was called to house to see patient and found marked swelling, as well as edema of right upper eyelid. Patient stated that she had consulted another specialist, who had advised an intranasal operation, to which she had consented, but as the swelling over frontal region had increased along with the appearance of the swelling about the eye, and some headache, which until now had been absent, she became alarmed, discharging her other specialist and then sending for me. On November 23rd, at my request, consultation was asked for, and a radical opera-

tion was again advised. On November 24th, she was taken to hospital with temperature of 99, pulse 100, respiration 24. Blood examination gave a leucocytosis of 9,600 with 72 per cent polynuclears. Spinal puncture showed fluid clear and normal and no tension. On November 26th, a radical frontal sinus operation was done. Upon opening into the frontal sinus, extensive necrosis in the anterior wall was found, together with a large defect in inner table. The dura was covered with granulation tissue, although not tense or bulging. The pus found in sinus produced a pure culture of hemolyticus streptococci. The wound was not completely closed externally, but lightly packed. On November 28th, patient complained of a severe headache and blood examination gave a leucocytosis of 11,600 with 82 per cent polynuclear cells, this being the highest count shown throughout the course until pneumonia developed. Temperature 99.2, pulse 124, respiration 24. Patient continued to improve and on December 11th was sent home. On December 15th, although very little pus and wound practically healed, patient complained of severe headache. This, however, was of short duration. On account of this headache and the previous attack of headache, along with the exposed dura, I advised an exploratory operation on frontal lobe for abscess, but as patient seemed to improve and headache disappeared, family would not consent to another operation. On December 19th, patient seemed very bright, no headache, temperature normal. On December 21st, patient developed pneumonia. On December 23rd, patient began with short periods of unconsciousness, from which she could be aroused and would answer questions. December 24th, found patient had developed paralysis of left side. Again advised reopening of frontal wound and exploratory operation of frontal lobe. This, however, was refused and patient died December 27th. Autopsy not granted.

From the report of this case it will be noted that although there was a considerable pathologic process in the early stage of the disease, which had gone on for some time, outside of an occasional headache, no intracranial symptoms presented themselves, and it was not until with the development of pneumonia that the other symptoms of frontal lobe abscess became evident.

With the above cases the diagnosis was easy, due to the knowledge of the original trouble combined with the symptoms of intracranial pressure. In the true frontal lobe abscess cases, symptoms are often very misleading or hidden and the diagnosis is generally difficult.

When considering intracranial symptoms arising from cases of frontal sinusitis, one must not lose sight of the fact that we are dealing with that portion of the brain known as the silent area, the functions of which are without marked motor manifestations.

Functions are practically established for the prefrontal lobe, and the second and third frontal gyri on the left side; the former having to do with acquired skill movements, and the latter with speech and expression. The rest of this region being the great motor association regions, any irritation or pressure on any given section of it may produce sensory disturbances, due to the close linking with the postcentral region through the associational tracts. On this account, through pressure arising from the vascular system engorgement, toxic irritation or malnutrition of the association fibres and cells, do we attribute our symptoms. This is most forcibly illustrated in Case 1, in which we have the paresis of the lower opposite limb, which immediately cleared up upon evacuating the extradural abscess. The return of the convulsions, due to the pent up pus, was followed by immediate relief upon evacuating same.

From a study of the two reports, you will note that the symptom of headache is most pronounced, being too severe to be accounted for by the sinusitis alone. One may be misled unless the severity of the headache is taken into consideration, as it is far more violent in the intracranial complications, a possible explanation being the congestion of the anterior meningeal blood vessels. The normal lymph flow and oxygenation to plasma and brain cells being interfered with by the congestion of the anterior cerebral, corresponding to the superficial congestion of the meningeals, the lack of oxygen results in demand by the cells referred to the thalamus and postcentral region and is projected back as pain. Extradural type of abscesses usually causes greater headache than intradural, due to the fact that we have, as a rule, a wider

involvement of the congested region. This is well seen in Case 1, where the original headache disappeared on free drainage of the frontal sinus, only to reappear in a more violent form, lasting until the epidural abscess was evacuated. In Case 2, the severe type of headache appeared late after our first operation. This, together with the appearance of the exposed dura, was the basis of our diagnosis of a cerebral abscess, at which time a second exploratory operation was advised.

In neither case was the pulse of any value, although it must not be overlooked. Many men call attention to it, laying great stress on the slow pulse, which indicates a general increased intracranial pressure.

Temperature, being one of septicemia, offers nothing characteristic. In the early stages of Case 1, relieved upon removing the source of trouble, while in Case 2, returning, when complicated with pneumonia.

In Case 1 patient complained of dizziness, which seems to be rather a rare symptom, while vomiting, although it did not occur in either case, is a great deal more frequent, and especially when occurring independent of meals, cerebral abscess must not be overlooked. This is due to the toxic or congestive irritation of the medulary center.

Referring to Case 1, it is well to note the marked symptoms of irritability and early convulsions, which at first were not localized. Disturbances of the sensorium are usually more or less marked, such as drowsiness, peevishness, weakness of memory, irritability, inclination to sleep, stupor, apathy, patient when questioned answers very slowly but correctly. These symptoms more or less marked, due to the degree of meningeal and cortical congestion.

In both cases, paresis of the opposite lower limbs was noted, Case 1 clearing up with the opening of the epidural abscess.

Optic neuritis and stasis of the papilla have been described in a number of cases, due no doubt to the increase of intracranial pressure, or possibly to the chronic passive congestion anaemia. This is well illustrated by the transient blindness which occurred in Case 1, although with the ophthalmoscope only a venous engorgement of the fundi was noticed. The external symptoms, or complications proceeding from the eyes,

are usually due to the sinusitis, but oculoorbital complications seem to appear more often in those cases which are complicated by intracranial lesions and, for that reason, acquire certain diagnostic value. This being due to the anatomic relationship of the lymphvenous flow from the orbit being inward, thus conditions affecting the intracranial blood vascular system would tend to produce similar changes in the orbit.

Spinal puncture in the first case showed an increased intracranial pressure, cell count and sugar, but no bacterial growth, while in the second case it was of little value. However, in looking through the literature I find that in those cases in which the puncture is made there is usually a slight increase of pressure and the presence of a copper reducing element. Owing to the various changes which take place the value of the spinal puncture can only be estimated to any degree by the condition of the patient.

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XXXII.

ACUTE MASTOIDITIS WITH UNUSUAL COMPLICATIONS.

By E. P. Norcross, M. D.,

Chicago.

The case that I wish to report this evening presented such unusual complications that I consider it worth while to review it before the Society.

While the history of the case covers a period of several months I shall make the recital of it as brief as possible, emphasizing the unusual features.

The subject of the report is a child three and a half years of age of unusual mentality. About four weeks before I first examined her she had contracted a head cold followed by a right otitis media purulenta. A paracentesis was done followed by a profuse mucopurulent discharge. Two weeks after the paracentesis the patient had a sudden rise in the temperature and complained of pain in her head and dysphagia. Examination of the throat revealed a retropharyngeal abscess on the right side which ruptured spontaneously before the doctor had time to incise it. The child's condition then improved until the time I first saw her, one month after the onset of the otitis media.

At this time the child had a temperature of 101.6, pulse 120. She was irritable and complained of pain in the right side of her head. When she moved she held her head because her neck hurt her. There was no neck rigidity, and with the exception of a paresis of the right external rectus, no evidence of paralysis anywhere. The reflexes were normal. There was a profuse purulent discharge from the right external auditory canal and well marked tenderness over the mastoid antrum and tip. The throat was clear. The tonsils had been removed the year before.

The decision to do a simple mastoid operation at once seemed undebatable. The antrum was full of pus which extended throughout the broken down cells forming a well

of pus at the tip. As the surrounding bone was hard, neither the lateral sinus nor the dura over the tegmen was exposed.

The child did not show marked improvement. The temperature became normal but the child did not regain her strength and continued to be irritable. The discharge from the external auditory canal was much less, but it did not cease. The wound did not show healthy granulations. Double vision and pain in the right eye were complained of for about a week. The W. B. C. remained at about 17,000 and the hemoglobin at 65; R. B. C., 3,500,000.

Ten days following the operation the temperature rose to 103 and the white blood count was 26,000. The child complained of pain in the neck and dysphagia. Examination of the throat disclosed another large retropharyngeal swelling, extending upwards into the postnasal space. Incisions into the mass resulted in the evacuation of about one-half ounce of thick, creamy pus. Cultures showed long chain streptococci which did not change blood. The W. B. C. dropped to 14,000.

Radiogram of the cervical spine did not show any disease of the vertebrae. The patient continued for another two weeks in fairly good condition. However, the discharge from the wound and external canal persisted. The W. B. C. again rose to 20,000.

Dr. Norval Pierce saw the patient with me and in view of the protracted course and the continued discharge the mastoid wound was reopened and a healthy dura and sinus exposed. As the cavum tympanum was filled with necrotic tissue the simple mastoid operation was converted into a radical operation.

Following this procedure the patient made rapid improvement, and was discharged from the hospital two weeks later. When last seen at my office the child was happy and good natured, submitted kindly to the ear dressing, and presented the picture of a healthy, normal child.

Three weeks later the mother telephoned to me and said that the child had become very angry about obeying some command and had had a violent spell of temper, screaming and kicking and scratching her mother. When she had been quieted, the mother had noticed that she did not seem able to

call familiar objects by name. Otherwise there was no change in her condition. A careful examination was made of the reflexes and eye grounds, but nothing unusual was discovered. However, she could not or would not tell the names of familiar objects, although she knew what their use was and recognized the name when it was spoken.

She was sent back to the hospital for observation. The temperature was not elevated, but showed a tendency to be subnormal. The pulse was occasionally retarded. At times the child complained of pain in her head, more on the left side.

About ten days after entering the hospital she began to show some weakness on the right side of the body, first the facial muscles and then the leg. The grip in the hands remained about equal. The eye examination showed a tendency to convergent strabismus and diplopia, slight bilateral papillitis. There was no increase in headache or speech disturbances.

The following day there was a noticeable weakness in the right arm and while I was talking with the patient she suddenly had a convulsion involving all the extremities. She did not regain consciousness.

A trephine opening was made over the left temporal sphenoidal lobe. The brain was bulging—incision into its substance was followed by a very copious discharge of thick pus. The patient died about fourteen hours later.

A postmortem examination of the brain showed the presence of two large brain abscesses. The older and outer one, situated in the temporosphenoidal and occipital lobes, measured 10 cm. anteriorly-posteriorly and 6 cm. wide at its widest part. It reached from a point 1 cm. in front of the optic chiasm to a point 4 cm. from the tip of the occipital lobe.

A second abscess smaller and more recent in origin was situated mesially to the first described abscess. A direct connection could not be found between the abscess cavities.

This case presents several interesting features. First the occurrence of a retropharyngeal abscess which undoubtedly was caused by an infection arising in the tubal tympani cells traveling along the eustachian tube under the mucous membrane or periosteum and perichondrium to the postnasal space. In a child this distance would not be great. I believe that

this is the correct explanation of the occurrence of the retropharyngeal abscess. There was no disease of the cervical spine, the throat was free from tonsils and adenoids, and the abscess ceased to form as soon as the middle ear was cleaned out.

I have not found the report of a similar case in the literature. The nearest approach is that of a case reported by P. Jacques, 1913. In this case during a mastoid operation necrotic area was found at the level of the floor of the antrum. A probe was passed about 1 cm. towards the lower angle of the sinus. Through this place enlarged by the curette an abundance of pus flowed during the following days without seemingly amending the phenomena of the general infection. There was a marked torticollis. The neck was painful upon pressure. Later dysphagia was complained of. An inspection of the throat revealed a mass in the right pharyngeal groove which, when incised, gave forth a large quantity of pus. From this day a new period began, characterized by a progressive decrease in the mastoid flow and of the phenomena of infection.

The second interesting feature is the development of a left temporal sphenoidal abscess during the course of rightsided otitis media. In reviewing the literature I have been able to find two somewhat similar cases. One reported by M. Lombard—Abscess of frontal lobe on opposite side from a chronic suppurative otitis. This patient was 28 years of age. Five years before he had been operated by Lombard for a chronic otitis media on the left side. On February 4, returning from work, the patient had an epileptic fit. The convulsions were the same on both sides; no tongue biting or emission of urine, eyes revulsed. The attack lasted five minutes. Amnesia followed. This fit was preceded for eight days by fatigue, and physical and mental depression.

February 9 he had another attack just like the first one. During the days that followed he complained of a continual headache, night and day, frontal and occipital; vertigo accompanied by nausea, both when standing and reclining; left ear painful with buzzing.

On February 14 patient in very marked stupor; no aphasia; no trouble in writing; read aloud in very loud voice; recited

the multiplication table; recognized objects and named them correctly, but with hesitancy. No olfactory trouble.

Operated under chloroform. Trepanation of the temporal fossa above and a little behind the external auditory canal. Incision of the dura mater. Hypertended cerebrospinal fluid escaped. Four punctures with a sound in different directions led to nothing. Dura mater sutured with catgut.

February 15. Left hemiparesis with hypoesthesia. On left side, Babinski in extension. Abdominal cutaneous reflexes disappeared. Cremasteric diminished, patellar greatly diminished. Stupor, slow idea formation, but no aphasia; no spontaneous nystagmus, no sensation of vertigo.

February 17. Hemiparesis persisting; slight stiffness of neck; no Kernig; no temperature.

February 20. Symptoms aggravated; patient almost in coma, incontinent, answers questions wrong. Conjugate deviation of eyes toward the right. No nystagmus; temperature 37. Second operation—a few whiffs of chloroform; left vertical retroauricular incision; treparation behind sinus; incision of dura mater. Escape of cerebrospinal fluid; repeated punctures of the cerebellum; dura mater sutured; wound tamponed with gauze.

February 21. Dies in coma.

Autopsy showed abscess of right frontal lobe as large as a large prune, encapsulated; pus of the abscess was sterile.

A second case is recorded by Klesseres—an abscess in the left brain with right otitis media. The right ear drum became red and the vessels of the fundus oculi a trifle distended. The reflexes of the right knee were increased—Babinski sign present—after three days paresis of the right arm was noted. Pulse 60—temperature normal. Three days later the left cortical center was exposed; an abscess was discovered 3 cm. in circumference and the patient died of pneumonia.

The diagnosis of the left brain abscess was based upon the symptoms of paralysis of right facial muscles and right arm. The most remarkable point is the occurrence of an abscess in the left hemisphere from a right otitis media.

The formation of a brain abscess by an infected embolus from some distant focus of infection is not so uncommon. The interesting feature in the cases here reported is that the

focus of infection happened to be an otitis media located on the opposite side of the head from the abscesses.

One cannot state in my case whether the child's fit of anger would favor the transmission of the embolus or whether there was present at the time of the outburst of temper a latent abscess that became active. Cases are reported of latent brain abscesses becoming active following some head injury or a mastoid operation.

Such cases as I have reported afford one material for serious thought and retrospection. It does not seem possible that an abscess of this magnitude could develop right under one's eyes, as it were, and not give definite enough symptoms upon which to base a diagnosis. There must have been a time during the course of the disease when an exploratory operation might have saved the child's life. Was the very mild aphasia in a right handed child a sufficient cause for doing such an exploration? Probably not, and yet I cannot help but feel that, had the otitis media been located in the left ear, the development of a mild aphasia would have been considered sufficient indication for reopening the mastoid wound and exploring the brain.

XXXIII.

THE ROLE OF THE TONSILS IN THE TREATMENT OF NOSE CONDITIONS, INFECTIVE AND OBSTRUCTIVE—STUDY OF 111 CASES.

By T. R. Gittins, M. D.,

Sioux City, Iowa.

The purpose of this paper is to report a series of 111 cases of nasal sinusitis or obstruction observed and treated during the past eight months. The object has been to trace the relation of the tonsils to the end results obtained in these nose cases. These patients have been under the care of two associates and myself in private practice. All were hospital cases.

The fact that tonsils do have a great influence upon nose conditions seems so generally conceded that very little is to be found in the literature on the subject except in the discussion of sinus disease in children. Dr. L. W. Dean,[1] of Iowa City, has done a great amount of work on the latter subject in the past few years, making exhaustive studies from many angles.

Granting as we must from our clinical observations that there is some relationship, both in adults and children, between the course of nasal infection and the condition of the tonsils, these questions arise: First—Does the presence of chronic tonsils predispose the patient to nasal infections? Second—Does the development of acute tonsillitis complicate the postoperative course of the nose condition and influence the end result? Third—Does the removal of tonsils only have a beneficial effect upon the nose infection at the time or render the patient less liable to develop future rhinitis or a sinusitis? Fourth—Does the presence or absence of tonsils have a bearing on the postoperative and end results in cases where the object was the removal of nasal obstruction only?

Before considering the case reports it is well to consult the anatomy of the nose and throat with reference to the course of infection, sinuses to tonsils, or the reverse. J. K. Milne Dickie[2] in an abstract raises the question whether in acute ton-

sillitis following nose operations the infection passes through the lymphatics, blood stream or by direct continuity. Schönemann held that the infection passed through the lymphatics. He injected a solution of iodin into the inferior turbinates, removed the tonsils on the same side six hours later and found iodin present. Lenart and Henke tried a similar experiment with dogs and came to the same conclusion. Auerbach following similar technic found none in the tonsils. Pugnat injected an emulsion of soot into the turbinates, 24, 36 and 48 hours preceding the removal of tonsils. In 100 such cases none of the soot was demonstrated in the tonsils. He concluded that there was no direct connection between the lymphatics of the nose and throat. Along similar lines W. V. Mullin and C. T. Ryder,[3] of Colorado Springs, have reported some exhaustive work in their studies on "Lymph Drainage of the Accessory Nasal Sinuses." They experimented with injections of India ink and bacilli in the sinuses of rabbits and cats. In summing up they say, "We were struck in the first series by the absence of any discoloration of the tonsils in animals in which nearby tissues were heavily injected, and drew the inference that the tonsil perhaps has no field of absorption beyond its own extensive surface." In two cases they injected India ink into the pharyngeal mucosa adjacent to the tonsils. Autopsy here showed marked discoloration in the tissues of the pharyngeal wall but no change whatever in the tonsils.

In discussing the development of acute tonsillitis following the submucous operation Loeb[4] expresses the belief that the main factor is the length of time that the packing is retained in the nose. He advocates the removal of the nasal plug within twelve hours to decrease the prevalence of these acute throat infections.

In this series of 111 cases treated because of symptoms of nasal obstruction or sinusitis, 53 were of the former type, 58 of the latter. Included in the obstructive cases are those in which septum or turbinates, singly or together, were responsible. Twenty-six of the sinus cases were classed as acute, 32 as chronic.

The entrance examinations of the 111 cases proved that 28 had had tonsils removed previously. In the 83 cases which

had tonsils present—I would prefer to call them all chronic tonsils, as who can say positively that any tonsil has not some infection—19 developed acute tonsillitis during the postoperative treatment. In 24 of the 83 cases the tonsils were removed at the time of the nose operation, thus eliminating this group from the possibility of acute tonsil infection. Here then, we have 59 postoperative nose cases, obstructive or infective with tonsils present, 19, or 32 per cent, of which developed an acute tonsillitis. As mentioned before in this paper, the theory of Loeb that acute tonsillitis develops in direct proportion to the length of time the packs are left in the nose may help to explain this high percentage. However, only in four instances did acute tonsil infection develop following the submucous resection or turbinectomy. There was no uniform method of packing adopted, the idea being to use the smallest amount of gauze or splint advisable and leaving it in place for the minimum length of time necessary to satisfactorily control the postoperative bleeding. Some of the cases had no plug, others had a small amount on one or both sides for a few hours, and still others were packed solidly on both sides for 24 hours. Certainly it is reasonable to expect that long continued packing of the nose will predispose patients to acute sinus or throat symptoms, but it is interesting and instructive to follow the even course of those cases in which the tonsils have been removed previously almost regardless of the length of time or completeness of the postoperative nasal packing.

In order better to tabulate results the submucous and turbinectomy cases are now segregated for study. There were 53 such cases in all, 34 of which had tonsils present on entrance examination. At the time of the nose operation the tonsils also were removed in 20 instances, thus leaving but 14 of the original 53 with tonsils present during the postoperative course. Four, or 28 per cent, of these 14 developed an acute tonsillitis during their convalescence. Loeb[4] quotes Horn[5] reporting a series of 110 submucous cases, 13 of which developed acute tonsillitis. One of these 13 in addition developed a severe pericarditis and endocarditis. No mention is made of the number of these cases in which the tonsils were removed previous to the nose operation. It is interesting to note here the length of time these various cases remained in

the hospital. The 4 which developed acute tonsillitis averaged 8½ days; the 10 with tonsils present, 3.7 days; the 19 with tonsils absent, 3.8 days, and the 20 in which the tonsil and nose operation was done at the same time, 37 days.

A goodly percentage of this group of cases was influenced to return frequently for examination, while in other cases we received some information from follow-up letters. Of the 4 cases in Group 1 (those which developed acute tonsillitis), 3 have been followed carefully, and all have bad results—that is, they were not only not relieved of their entrance complaints but were distinctly worse than before their operation. These cases will be considered later more in detail. Of the 10 cases in Group 2 (those with tonsils present), 4 have not been heard from; 1 has a good result after six months; 1 after three months; 3 after three weeks, and 1 continues to have some difficulty in breathing and some discharge from the nose after four weeks. There were 19 in Group 3 (those with tonsils absent), with the following statistics: Four have not been heard from; 1 has a good result after six months; 3 after four months; 4 after three months; 2 after two months; 3 after one month; and 2 after two weeks. In Group 4 (those with tonsils removed at the same time as the nose operation), we find the following: Seven have not been heard from; 2 have a good result after three months; 3 after five weeks; 2 after three weeks; and 6 after two weeks. In the above statistics by good results is meant relief from the symptoms of which the patient complained on entrance examination and the development of no new symptoms. It is quite likely that a goodly percentage of those cases not heard from have a good result or we would have at least had complaints.

Returning to Group 1, we will consider these four cases in more detail.

Case 74 had a partial turbinectomy, middle each side. Two days later developed a mild tonsillitis with temperature of 99.6. No report has come from this patient after she left our care.

Case 19.—Mr. M., age 23, entered complaining of difficult breathing through nose, of long standing. No history of head colds or tonsillitis. X-rays of sinuses were negative. His diagnosis was deviated nasal septum and chronic tonsillitis. Tonsillectomy was advised but refused. On March 9th a sub-

mucous resection of the septum under local anesthesia was done. Course uneventful until March 13th, when he developed sore throat, temperature 102.6, membrane on tonsils. On the second day, or March 15th, there was much swelling of the turbinates and septal flap, thin pus was present on each side of the nose. Patient left hospital on March 17th, eight days after entrance. His general condition remained poor. He looked anemic, had poor appetite and tired easily. There continued a free discharge of pus from each side of the nose, and the swelling of the septal flap and turbinates subsided very slowly. Transillumination of antra was positive. X-ray of sinuses was refused. On April 9th, or one month following submucous operation, there was still free discharge of pus from each side of the nose, a perforation of the septum and boggy inferior turbinates. His appetite and strength were below normal and he was unable to carry on his ordinary work. When last seen, two months after his operation, he still had difficulty in breathing through his nose, much discharge back in the throat, and a chronic sore throat.

Case 30.—Mr. S., age 28, entered hospital complaining of difficult breathing through nose, no sore throat, occasional head colds. Diagnosis was deviated septum and chronic tonsillitis. Tonsillectomy was advised but refused. Submucous resection under local anesthesia was performed March 29th. March 30th, temperature was 102.4. Next day membrane over each tonsil. Fever remained above 100 for six days. Septal flap and turbinates badly swollen, almost completely obstructing nasal passages, much fullness and headaches in frontal region. Left hospital after seven days. Patient next seen in two months; still having difficulty in breathing through nose, much thick mucus from nose and back into throat. Had had some sore throat all the time since last seen. Tonsillectomy May 17th. Patient left hospital May 19th. To date has not been heard from.

Case 40.—Mr. W., age 36. Entrance complaint, difficulty in breathing through nose, frequent head colds in winter time. No sore throat or ear symptoms. Diagnosis, deviated septum and chronic tonsillitis. Tonsillectomy advised but refused. (It is our practice to advise the removal of tonsils before or at the same time in all cases where the submucous operation

is performed. The probability of acute tonsillitis developing and the dangers of same are explained to the patients and relatives.) May 2nd, submucous resection under local anesthetic. March 7th, patient left hospital with septal flap healing nicely. May 9th, developed acute tonsillitis. May 10th, considerable swelling of the septal flap and inferior turbinates and some thick mucus in the nose. May 11th, developed acute otitis. Paracentesis was done. A week later ear was discharging pus freely. There was still considerable swelling of the septal mucosa and turbinates and thick mucoid discharge from each side of the nose. At this time patient passed from under our care and has not been heard from since.

Of these four cases where acute tonsillitis developed, three were followed long enough to convince us that they were not only not benefited but were distinctly worse than before they came under our care. Cases 19 and 30, with negative histories and X-rays previous to the attacks of tonsillitis, have had free discharge of pus from each side of the nose following the acute infection. Both patients refused another X-ray of the sinuses, but in Case 19 both antra were very cloudy to transillumination. Case 40 had had no previous sinus or ear symptoms, but following the attack of tonsillitis had pus in each side of the nose and an acute otitis.

Before passing from the submucous cases it is well to note that in 20 of the 53 cases the tonsils were removed at the same time the nose operation was performed. In three of these twenty the combined operations were done under local anesthetic, the remaining seventeen under ether anesthesia. Especially noticeable was the lack of shock in those patients operated under general anesthesia and the rapidity with which the nose and throat healed. Naturally, greater difficulties were encountered in doing the submucous work under general anesthesia than under local, but we feel that with care good work can be done with the former method.

Continuing the study of the cases, we find that 48 of the 111 considered in this report were operated for relief of sinus disease. Of these 48, 20 were antrum cases, 10 frontal, 17 pansinus and 1 sphenoid.

The antrum cases will be taken up first. On entrance to the hospital 16 of these 20 had tonsils present. In 4 the tonsils

had been removed previously. Of the 16 with tonsils present, 7, or 43 per cent, developed an acute tonsillitis in the postoperative period; 4 were of chronic, 3 of acute sinus involvement. Eleven of the 20 antrum cases were seen in the acute stage, 9 in the chronic.

Looking at these statistics from another angle, we find that of the 11 cases which came under our care because of acute antrum disease only 1 had had the tonsils removed previously, the other 10 suffering from chronic tonsillitis at the time the acute sinus disease developed. Of the 9 cases with chronic antrum infection 3 had had the tonsils removed previous to our examination. In these latter cases, however, we have no way of determining just when the chronic sinus disease began. Might it not be possible that the chronic antrum infection was present some time before the tonsils were removed? It seems significant that only one acute antrum infection came under our care in a period of eight months, in a patient whose tonsils had been removed previously, while during the same period ten patients with a similar acute infection entered, each suffering from coincident chronic tonsillitis.

Passing on to the frontal sinus cases we find that they were 10 in number. Six of these were seen in the acute stage, 4 in the chronic. Of these 10, 8 had tonsils present on entrance examination. In contrast to the antrum cases, only 1 of the 10, or 12 per cent, developed acute tonsillitis, as against 43 per cent when we were dealing with the antrum. Of the 3 chronic frontals with tonsils present, 1 developed acute tonsillitis. Of the 5 acute frontals with tonsils, none developed acute tonsillitis.

In grouping the pansinusitis cases, we find there were 17. Seven of these were of the acute, 10 of the chronic type. On entrance, 14 of the 17 had chronic tonsils present, almost duplicating the antrum statistics. Six of this number, or 42½ per cent, developed acute tonsillitis during the postoperative course. These 6 cases were divided equally between the acute and chronic types.

Comparing with the antrum and frontal statistics, we find that of the seven patients treated for acute infection of all the sinuses on one side, all had chronic tonsils. In other words, no acute pansinus case was encountered where the patient had

had the tonsils removed previously. On the other hand, of the 10 chronic cases 3 had had the tonsils removed previous to our examination.

Only one isolated sphenoid infection came under our observation in this series of cases. This was of the acute type and had chronic tonsils present. Two days after the sphenoid was drained, this patient developed acute polyarthritis. Sinus was clear in eleven days.

Summing up, it may be significant to note that of the 24 patients who entered our services suffering from acute sinus disease, only two of the number had had the tonsils removed previously. Does this indicate possibly that patients without tonsils are more likely to escape acute sinus involvement or are we to assume that the ratio of patients with tonsils to those without is as 22 to 2?

TABULATION: SINUS CASES.

Case No.	*Type	DAYS UNDER TREATMENT — Acute: Complicated by Tonsillitis	Acute: Chronic Tonsils	Acute: Tonsils Absent	Chronic: Complicated by Tonsillitis	Chronic: Chronic Tonsils	Chronic: Tonsils Absent
1.	F				60		
2.	A					60	
4.	F			9			
5.	P	40					
6.	F						
7.	A						90
8.	F		15				
9.	A	30					
10.	A					90	
11.	A						20
12.	P					60	
15.	P		60				
16.	A					45	
18.	A			6			
20.	A						120
22.	A		15				
23.	A		30				
24.	F		21				
25.	A		24				
26.	F		15				
27.	A				21		
29.	A		14				
31.	P		15				
33.	F				30		
34.	F		10				
35.	P				45		
37.	A				45		
38.	A		8				
39.	P		90				
44.	P					120	
45.	P				80		
47.	P	130					
48.	P		14				
51.	P						14
52.	A	28					
53.	A		12				
59.	P	60					
61.	F		14				
62.	F						15
64.	P					60	
65.	A	45					
66.	P					60	
69.	P						45
87.	P						90
88.	P				180		
90.	A				30		
110.	A						
Average Days		55	23½	7½	61	70	56
No. of Cases		6	15	2	8	7	7

*F—Frontal; A—Antrum; P—Pansinus.

In a study of the above table we find a great similarity in the number of days under treatment in the chronic cases, regardless of the presence or absence of chronic tonsils or the development of an acute tonsillitis in the postoperative course. On the other hand, there is a wide variance in the statistics of the acute cases. Two patients with no tonsils present developed an acute sinus disease, and the average length of time under treatment was 7½ days. The 15 acute cases with chronic tonsils present averaged 23½ days of treatment, while in the six which developed acute tonsillitis the average time treatment was continued amounted to 55 days. These statistics suggest to us at least that where we are dealing with acute sinus disease in the presence of chronic tonsils, we may reasonably expect a longer convalescence. If acute tonsillitis comes as a complication, we may find an originally acute case becoming chronic, even when the drainage is adequate and irrigations frequent.

As was done in the submucous cases, a few of the interesting case histories will be cited in more detail.

Case 2.—Mr. C., age 35. Came to the medical service with an acute arthritis. Some pain over right face, tenderness over antrum, right antrum dark to transillumination and blurred in X-ray. Chronic tonsillitis. Teeth negative. Right antrum was drained intranasally January 5th, local anesthesia. Large quantity of thick old pus was washed out. Sinus was irrigated twice daily thereafter. On January 8th, or seven days after antrum operation, patient developed an acute tonsillitis. On January 10th, complained of pain over left antrum and a recurrence of pain in the right knee and neck muscles. On this date thick pus was irrigated from the left antrum. Five days previously this antrum was entirely negative. Patient gradually improved from this time on, but there was still considerable pain and stiffness in the joints and some pus in the antra. On January 21st, still some pus in each antrum, patient confined to bed because of arthritis. On this date tonsillectomy was done, local anesthesia. Twelve days following this operation both antra were free from pus and joints had cleared so rapidly that the patient was allowed to leave the hospital. Patient was next seen on July 5th, when he reported that all

joints and muscles were feeling as good as ever. He had no discomfort. Both antra were clear at this time.

Case 5.—Mr. K., age 45. Diagnosis on January 20th, acute frontal sinusitis, right. Diseased stumps of tonsils. Right antrum negative. On this date anterior ethmoids were curetted and right frontal drained. Patient continued to have some pain over frontal sinus, some pus in nose. On January 27th, developed an acute tonsillitis. January 30th, pain over right side of face, right antrum dark to transillumination. February 1st, right antrum drained; much thick pus present. For seven days following the draining of the acute frontal in this case the antrum on that side remained clear. Three days after acute tonsillitis developed this same antrum was full of pus.

Case 37.—Mrs. R., age 29. Diagnosis, sinusitis antral acute, bilateral with otitis media supp. acute left. Chronic tonsillitis. April 10th, both antra drained intranasally. Much pus found in each side. Drum membrane incised. April 18th, still small amount of pus in each antrum. No discharge from ear. April 20th, developed acute tonsillitis. On the next day there was a recurrence of pain in the ear and it began to discharge. April 23rd, much pus in each antrum and a free discharge from the ear, temperature normal; membrane has disappeared from tonsils. April 23rd, tonsillectomy. April 27th, ear again dry. May 11th, no pus in either antrum and no return of earache or ear discharge. Patient left hospital at this time.

Case 45.—Mrs. M., age 40. Diagnosis on May 21st was ethmoiditis chronic bilateral and empyema chronic left antrum. Chronic tonsillitis. On that date ethmoids on each side and left antrum were drained intranasally. There was much pus in the antrum. May 25th, antrum clearing rapidly, only a small amount of discharge. May 26th, patient developed acute tonsillitis. Two days later antrum was again filled with thick pus. Patient was again seen on June 15th. At this time a large amount of old pus was irrigated from the antrum. When patient was last seen, on September 8th, there was still pus being irrigated from the antrum.

Case 52.—Mrs. R., aged 28. Diagnosis on June 4th, empyema acute, each antrum. Chronic tonsillitis. Antra were drained intranasally. On June 9th, developed acute tonsil-

litis; slow convalescence, with a gradual decrease in the amount of pus in the antra. On June 30th, antra clear.

Case 65.—Mrs. L., age 32. Diagnosis on July 11th, empyema acute right antrum. Antrum was washed out through a small trocar daily for four times. There was now no pus, so patient returned home. On July 18th, patient returned to the hospital with an acute tonsillitis. Two days later developed pain over the right antrum, and on irrigation much thick pus was found. July 21st, developed an acute suppurative otitis right. July 25th, mastoidectomy simple right and intranasal drainage of right antrum. August 25th, mastoid cavity almost healed, ear canal dry, still a small amount of pus from the right antrum. Tonsils remained subacutely inflamed. September 27th, mastoid wound healed and antrum clear. October 2nd, tontillectomy.

Case 66.—Mrs. H., age 56. Diagnosis on July 23rd, pansinusitis chronic bilateral with polyps; chronic tonsillitis. All sinuses were drained intranasally on this date. Much old pus found in each antrum. Antra were irrigated once daily, and on September 7th there was still a small amount of pus in each one. Nose was quite clean and there was a good breathing space. September 8th, developed an acute otitis media right. September 26th, still thick pus from the right ear canal, small amount of pus from right antrum. Left antrum clear. September 28th, developed acute tonsillitis. Three days later there was much pus in each antrum. At the time this record is written, October 25th, there is still much pus in each antrum.

Case 88.—Mr. S., age 26. Diagnosis on entrance, sinusitis ethmoidal and antral, chronic bilateral. Chronic tonsillitis. All sinuses left were drained intranasally. Sinuses right were not drained because patient objected. After two months of daily irrigation there was still much pus in left antrum. At this time developed acute tonsillitis. For two months both antra were irrigated practically every other day and there continued to be pus present. Tonsillectomy was done. Thirty days after removal of tonsils no pus in either antrum, nose clean. Patient has not returned since that time. It is well to note here that the sinuses on the right side were never drained except the preliminary irrigation for diagnosis, yet in

thirty days after the tonsillectomy there was no pus present on either side.

Having considered the cases in which the submucous or sinus operations were done, there is left only a small group of 10 of the original 111. Three of these 10 passed from under observation before complete examinations could be made. The seven remaining patients were those with nose symptoms—that is, frequent head colds, poor nasal breathing, etc., in which only the tonsils were removed. Any nose treatment was either not advised at the time or refused by the patients. The small number of cases in this group is due to a large extent very likely to the fact that in the series of 53 submucous and turbinate cases the tonsils were removed at the time of the nose operation in 24 instances and within two weeks prior to or following on eight occasions. Thus in seven of the twenty-one remaining cases the tonsils only were removed in an attempt to relieve the nose symptoms. Of these seven, from three has come no report since their operation.

Case 94.—Mr. C., aged 34. Two years previous to examination had had middle turbinates trimmed and ethmoids drained. At the time of our examination the patient still complained of head colds and frontal headaches and occasional acute tonsillitis. March 7th, tonsillectomy was done. On September 5th, or six months later, patient reported that he had been very free from head colds or headaches. He says that his nose has given him less trouble than at any time in his life before.

Case 95.—Mr. P., age 38. Had submucous resection four years previous to our examination. Still complained of frequent head colds, poor nasal breathing and some sore throat. Examination reveals straight septum and good breathing space; chronic tonsils. Tonsillectomy, January 3rd. Patient was again seen on September 12th, or practically eight months later, and reported that he had been almost free from head colds and nose symptoms since the tonsillectomy.

Case 99.—Sister G., age 43. Entrance complaint, catches cold easily, frequent sore throats and cough. Diagnosis, deviated septum, chronic tonsillitis. June 5th, tonsillectomy. Patient was next seen September 14th, or practically eight months later, and reported much improved. She said there had been

very few colds, much less nasal discharge and that she felt so much relieved that she did not wish the nose operation performed.

Case 36.—Baby O., age 2½ years. Diagnosis, otitis media suppurativa chronic left. Chronic tonsils and adenoids. There was much pus in each side of the nose. Adenoids were removed. Patient was not seen again for three weeks. Then there was in addition to the chronic otitis an acute otitis in the other ear. There was still much pus in the nose. At this time tonsils were removed. Six days after the tonsillectomy the acute ear was dry and there was much less pus in the chronic ear. Fourteen days following tonsillectomy both ears were dry and the nose was quite clean.

No further mention will be made of the sinus cases in children except to refer again to the work of Dr. L. W. Dean[1] of Iowa City. He states that 80 per cent of the chronic cases of nasal sinus disease in infants are cured by the removal of tonsils and adenoids. He attributes these results to the age of the patients, excluding long chronicity, and to comparative infrequency of nasal obstructive lesions in infancy and early childhood. So firm is his belief in the efficacy of removing tonsils and adenoids that even where pus is demonstrated in the sinuses, he practically always attends to the throat condition first, then has the patient return in a few months for further examination before any treatment of the sinus condition is advised From this method of procedure he has deducted the percentage of cures given above. To illustrate, he cites the case of an eighteen-months-old baby with a posterior cleft palate. Adenoids were removed and careful irrigation of the nose was carried out for two months. They were unable to clear the patient of a hemolytic streptococcus sinus disease. Tonsils had not been removed, because of the fear of contraction of the pillars interfering with the success of the cleft palate closure. Finally the tonsils were removed, the sinus disease disappeared rapidly and the cleft was readily closed.

SUMMARY.

1. One hundred and eleven nose cases were studied. The diagnosis in 53 instances was deviated septum or enlarged

turbinates. In 58 cases it was sinusitis. Of the sinus cases 26 were treated during the acute stage, 32 during the chronic.

2. Twenty-eight of the 111 cases had had the tonsils removed previous to entrance examination. In 24 of the remaining 83 the tonsils were removed at the time of the nose operation. Thus 59 of the original 111 patients had tonsils present during the postoperative course. Of these 59, 19, or 32 per cent, developed an acute tonsillitis following their nose operation.

3. In the 53 submucous or turbinectomy cases, 4 had tonsils present on entrance examination. In 20 instances the tonsils were removed at the time of the nose operation. Thus only 14 of the original 53 went through their postoperative course with tonsils present. Of these 14, 4, or 28 per cent, developed an acute tonsillitis

4. In 3 of the 53 submucous or turbinectomy cases the end result was poor—that is, the patients were not only not relieved of the symptoms complained of on entrance examination but were distinctly worse than before the operation. In these three instances acute tonsillitis developed during the postoperative course.

5. Uniformly good and rapid results were obtained in cases of submucous resection where the tonsils had been removed previously or at the time of the nose operation.

6. Only two cases of acute antral and none of acute frontal sinusitis were encountered in patients whose tonsils had been removed previously. In this series acute sinus disease appeared almost always in patients suffering from acute tonsillitis.

7. In several instances acute antra developed following acute tonsillitis, complicating the course of the nose operation where the antra were clear previously. On the other hand, several acute antra resisted very active treatment until the tonsils were removed, then cleared up rapidly.

8. No frontal sinusitis case developed acute tonsillitis during the postoperative course.

9. Comparing the length of time of after-treatment in acute sinus cases, the two without tonsils averaged 7½ days, the five with tonsils averaged 23½ days, the six which developed acute tonsillitis averaged 55 days.

10. The chronic sinus cases remained under treatment practically the same number of days, regardless of the presence or absence of chronic tonsils or the development of an acute tonsillitis.

11. Quoting from Dean's statistics, probably 80 per cent of the sinus cases in infants and children are cured by the removal of tonsils and adenoids only.

12. In ten cases tonsils only were removed for the relief of nose symptoms. Four of these ten have been followed for a period of six to eight months and all have been greatly improved.

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XXXIV.

SUPPURATIONS OF THE LABYRINTH.

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HISTORY.

The development and study of labyrinthine suppuration, from the earliest experiments to the scientific studies of the present day, is a most interesting work. The studies of Flourens in 1824 to 1828 and followed later by the experiments of Ewald were the foundations upon which our present day literature is founded. Later several noted scientists, among whom are Jansen, Hinsberg and Richards, added greatly to the subject. The name of Robert Barany of Vienna perhaps stands foremost among the investigators of labyrinthine suppurations. He has conducted many experiments and has given much valuable time to the study of the labyrinth. The works of Neuman and Ruttin followed, and in this country much credit is due to further scientific study of this topic by such men as Kenison, Ballenger, Mackenzie, Friesner, Dench, Shambaugh and Duel.

ANATOMY.

That we may understand this subject better, perhaps a brief outline of the anatomy of the labyrinth would not be out of place.

The inner ear is composed essentially of two structures, (1) the osseous labyrinth, and (2) the membranous labyrinth. (1) The osseous labyrinth is made up of a series of cavities continuous with each other, namely, the cochlea, vestibule and semicircular canals. The vestibule makes up the central portion, communicating posteriorly with the semicircular canals and anteriorly with the cochlea. Posteriorly are the five apertures of the semicircular canals. Anteriorly the vestibule opens into the scala vestibuli of the cochlea.

The three semicircular canals lie posteriorly and above the vestibule, being differentiated by position, namely, the superior

or anterior vertical, posterior and external or horizontal. They open by five apertures into the vestibule. The inner extremity of the anterior vertical and the upper extremity of the posterior unite and form one canal opening into the vestibule.

The cochlea is a cone shaped structure, having the base corresponding to the internal auditory meatus and apex pointing outwards and forwards. It is a sort of spirally arranged tube surrounding a central portion, the modiolus. The lamina spiralis ossea is a thin bony process which divides this spiral tube structure into two parts, the one above called the scala vestibuli and the one below the scala tympani.

(2) The membranous labyrinth, lying within the bony labyrinth, contains a fluid called endolymph, and the space between it and the real bony labyrinth is called the perilymphatic space, which contains perilymph. The utricle and saccule are the two membranous sacs in the vestibule. The former receives the endolymph from the membranous canals and the latter communicates with the membranous cochlea. All portions of the membranous labyrinth contain endolymph and communicate with each other. The ductus endolymphaticus, from the saccule uniting with the ductus utriculosaccularis from the utricle, are transmitted by the aqueductus vestibuli to communicate with the meningeal lymphatics. The inner layer of this membranous labyrinth throughout is composed of a supporting layer of cells, upon which rest the hair cells proper.

EXPERIMENTS OF EWALD.

From Ewald's experiments the following deductions are made:

(a) Excitation of a single canal produces a nystagmus only in a plane that corresponds to the plane of the canal.

(b) By reverse movements of the endolymph in any canal the induced nystagmus is reversed.

(c) The strongest nystagmus which can be induced by irritation of a single canal is always in the direction of the ear experimented upon.

A nystagmus of the vestibular origin has the following characteristics:

1. Is composed of a quick movement (quick component) in one direction and a slow movement (slow component) in the opposite direction.

2. When the eyes are turned voluntarily in the direction of the quick component, the nystagmus is increased in rapidity and in length of excursion.

3. On turning the eyes in the direction of the slow component, the nystagmus becomes very weak and at times disappears. These characteristics are usually to be elicited during the course of any acute suppurative labyrinthitis.

THE CALORIC REACTIONS.

To Barany of Vienna belongs the honor of establishing the significance and the technic of the caloric reactions and of the conclusions drawn. They are dependent on the following conditions:

1. No objective or subjective phenomena result when a normal ear is irrigated with water at ordinary body temperature.

2. Irrigating an ear with water at 70 to 78 degrees F. the following phenomena occur:

(a) A rotary nystagmus to the opposite side (towards the ear not irrigated).

(b) Patient becomes dizzy and at times nausea and vomiting may set in.

3. Irrigating an ear with water whose temperature is 110° we note the following phenomena:

(a) A rotary nystagmus to the same side (toward ear irrigated).

(b) Ataxia and vertigo and if severe enough and nausea and vomiting. I have seen patients become very much nauseated and yet never reach the vomiting stage.

The real value of all these caloric experiments is that we can determine to what extent, if any, the vestibular loss has been. This, of course, depends on the fact that if the vestibular apparatus has been impaired or destroyed, we find no caloric reactions on applying water whose temperature is 78 or 110 degrees (cold or hot).

By placing the head in the different positions one is able to thus test each semicircular canal and note its normal physiologic action.

There are two tests that stand out foremost in determining functional activity of the labyrinth. The noise apparatus given us by Barany, which enables one to determine partial or complete loss of hearing, and the caloric reaction test which reveals to us the presense or absence of static function. It is quite necessary in order to obtain true findings in the caloric reactions to have the canal free from any material, such as cholesteatomata or granulations that would inhibit the proper action of the water used. The turning tests, which are of value in certain cases, are to be used with just care, for no one knows how many cases which were in a fair degree becoming localized were by those tests converted into a diffuse suppurative condition.

The spontaneous nystagmus is really a manifest sign of acute labyrinthitis, and when the slow component is directed towards the sound side it means an overstimulated labyrinth, while if directed towards the diseased side it means either a weak or absent function. The vestibular apparatus is responsible for the slow component of the nystagmic movement.

Another important reaction, though when only positive, is the fistula test. This test should only be made when the caloric reaction appears negative. When the fistula test is positive so also one generally finds the caloric test positive. A positive fistula symptom reveals to us that the inflammatory reaction has already invaded the membranous labyrinth.

While performing a radical mastoid operation, if a fistula presents itself, it is the concensus of opinion not to probe it where there is absence of meningeal symptoms. For to do so would tend to light up a quiet process, converting a perilabyrinthitis into an active diffuse affair.

If the caloric test is positive it is better to wait until time of operation to discover the fistula. In an acute labyrinthine case, if it is determined that there is a dead labyrinth by a negative caloric and ablated hearing, it is also considered to be unwise to subject the patient to the other tests, such as whirling, turning, standing, etc. Absolute quiet is the rule in order to keep the inflammatory reaction circumscribed and to prevent the extension of same to the meninges. Nature has always been very kind to us, and here in this location she has thrown up an inflammatory wall between the meninges and

the labyrinth, consequently it is to the welfare of our patient to preserve its presence.

ETIOLOGY.

As is well known, the etiology of suppurative labyrinthitis is very rarely primary. It is usually secondary to diseased conditions of the middle ear, especially otitis media suppurativa, which so frequently follows the acute exanthematous fevers, as measles, mumps and scarlet fever, particularly the latter. Suppurative labyrinthitis may follow pneumonia, meningitis, and even cases of thrombus in the petrosal sinus of the temporal bone. Undoubtedly a suppurative process might be set up as a result of meningitis, the infective agent gaining access through the lymph channels, the aqueduct of the vestibule and the cochlea. Only occasionally are suppurative processes set up in the labyrinth as a result of acute otitic infections; more commonly they occur as a sequence of long standing middle ear suppuration. They occur very frequently as an acute exacerbation of a chronic process of tympanic suppuration.

CHANNELS OF INFECTION FROM MIDDLE EAR TO LABYRINTH.

The labyrinth may become infected systemically through the blood vessels and the lymphatics, and indeed, many of those unexplainable inflammations are in all probability due to such channels. Shambaugh[1] has worked out the circulation of a guinea pig and has demonstrated that there is quite a definite anastomosis of the blood vessels of the labyrinth and those of the middle ear. It must also be remembered that toxins of a systemic disease may become localized in the labyrinth, being carried by the vessels, and thus a localized labyrinthitis becomes apparent, and soon, by lowering the vitality of the structure, makes it a ready playground for the many pyogenic bacteria which are constantly in the circulation.

There are no direct communications of the middle ear and the labyrinth. The two openings present in the osseous bony structure, foramen rotundum and foramen ovale, are covered by a dense tough membrane which stops the progress of all germs. Many different accidents may occur to cause a perforation of the outer labyrinthine wall and thus afford an easy

channel of entrance to the vestibule of an existing middle ear trouble. In cases of fracture of the base of the skull through the petrous bone, microorganisms may invade the labyrinth from the nasopharynx by way of the eustachian tube and also externally through the auditory canal. During the operative attempt of extracting the stapes when necrotic, especially if in unskilled hands, the labyrinth becomes infected. Even in the removal of foreign bodies from the ear, the stapes has become dislodged and the labyrinth infected. Also during the radical mastoid operation, great care must be exercised in removing the malleus and the incus, lest the stapes should be pulled away and the membrane covering the foramen ovale become perforated.

Victor Hinsberg[2] of Breslau, after carefully searching the literature for labyrinthine suppuration complications of the middle ear, found only 198 cases which had been carefully recorded. The entrance of the infection from the middle ear to the labyrinth could be determined in only sixty-one cases of the 198. Below are given the locations as he reports them:

Fistula in horizontal semicircular canal	27 cases
Fenestra ovalis	17 cases
Fistula in posterior or superior semicircular canal	7 cases
Fistula on promontory	5 cases
Fenestra rotunda and ovalis	3 cases
Fenestra rotunda	2 cases

From the above statistics, it will be readily noticed that the infection entered the labyrinth most frequently through erosion of the horizontal semicircular canal, then through the oval window, the infection gaining entrance in only two cases through the round window.

The reason for the frequency with which the horizontal canal is involved can be ascertained on studying this from the anatomic point of view. We find

1. The horizontal or external semicircular canal protrudes somewhat upon the floor of the aditus, like a small rounded eminence.

2. Being situated in this way at a point of greatest constriction between the antrum and the tympanic cavity, it is

subject to the action of all the suppurative process of the middle ear.

3. The aditus is made up of a hard, dense, bony ring. The one exception is the horizontal canal, and this represents the weakest spot in this bony ring.

PATHOLOGY.

The pathology of suppurative labyrinthitis consists in finding the inflammatory products in the endolymph, and these may be localized or diffused throughout the vestibule and all the canals. The fibrin becomes organized, a vascularized connective tissue is formed around the entrance of the infection. If of a very virulent infection the process becomes quite extensive, and granulations may or may not be formed in the acute form. In the chronic form or where a labyrinthine infection has been set up following a long history of otitic trouble, a great many small granulations are found. Some of the canals on being opened, especially the external semicircular canal, are seen to be practically occluded by granulation growth. This vascularized connective tissue may in time be transformed into a hard, bony substance by the deposits of calcium salts. Much of the labyrinthine wall may become absorbed and broken down. Microscopically, round cell infiltration is found among the granulations, the serous membrane lining the vestibule, and the canals become congested and swollen; round cell infiltration is found in most of its walls. The footplate of the stapes may be necrosed and only a small portion left, or it may be embedded in the surrounding exudate. In one case, on opening the first and second turns of the cochlea, as is always performed in a complete labyrinthectomy, a purulent exudate was readily seen exuding from the opening. Politzer[3] states that in some cases the absorption has gone on to such an extent in the cochlea that only a small fragment of one turn was remaining and the modiolus was almost wholly gone.

The terminations of infective labyrinthitis from a pathologic viewpoint may be as follows:

(a) Complete return to the normal. The drainage being sufficient to allow most of the infective process to drain away. These cases are rare.

(b) The labyrinthine cavity may become filled by fibrous tissue. The numerous granulations present soon become permeated by connective tissue structure, and the many fibrous bands present tend to make it a hard, compact mass, which soon contracts and, in time, by deposition of calcium salts, be transformed into bone. The function of the labyrinth is much diminished in such cases.

(c) Extension of the infection to the meninges, through the aqueducts of the cochlea or vestibule; internal auditory meatus or by osteitis of the capsule from the superior semicircular canal; formation of extradural abscess, thence meningitis.

The fistulous openings in the external capsule, which are frequently found at operation or at postmortem, probably do not always represent real avenues of invasion from without inward, but at times avenues of escape through which a serous or purulent exudate is successfully discharged from the labyrinth into the tympanic cavity.

CHANNEL OF INFECTION FROM LABYRINTH TO BRAIN.

The cranial cavity frequently becomes infected by extension of the process along the acoustic nerve and the aqueducts of the cochlea and the vestibule. The infection may also extend through the internal auditory meatus, the latter being a means of common invasion. Anatomically speaking, any infection of the labyrinth may take place easier from the cranial cavity through the aqueducts of the cochlea and vestibule and through the internal auditory meatus than coming from the middle ear. For here the infection must invade and break down membranes and bones—in fact, overcome anatomic barriers to reach the labyrinth, while an infection from the dura simply has to follow well defined pathways which lead directly to the labyrinth.

SYMPTOMS.

In an acute suppurative involvement of the labyrinth the first symptoms to arise are those occurring as the result of irritation of the end filaments of the cochlear and vestibular nerves. The former by impairment or loss of hearing and tinnitus aurium, and the latter by loss of equilibrium, vertigo, nausea, vomiting and nystagmus. If the patient is not already

confined to bed this forces him to go. The patient will complain of a peculiar vertigo in that all objects tend to rotate before his eyes.

A nystagmus of vestibular origin is composed of a slow movement in one direction and a quick movement in the opposite direction and rotary in character. The slow component, at the first invasion of the labyrinth, will be directed to the sound side, showing an increased functional activity of the vestibular apparatus involved. In a short time (three to four days, depending on the virulent character of the infection), the slow component will be directed to the diseased side if the vestibular apparatus has been destroyed. If the slow component is directed to the sound side it is considered evidence of some function remaining in the vestibular apparatus and would also indicate, in a slight degree, that the deafness was not entirely lost.

The nystagmus to the sound side is explained upon the fact that when the labyrinth has been destroyed on one side there is an overstimulation of Deiter's nucleus on the opposite side and hence a nystagmus to the sound side follows.

Usually the first symptoms to appear are dizziness, subjective noises and spontaneous and these in turn are the first to go, while the disturbance of equilibrium, if present, will continue for a variable time.

Kopetzky[4] holds that in labyrinthine infection the onset is found to be marked with a distinct rise in temperature and sudden appearance of facial paralysis. On the contrary, Dench holds that facial paralysis does not occur early or is a common sign of purulent labyrinthitis. Facial paralysis usually occurs in cases of long standing necrosis. As to the frequency of facial nerve involvement, many different reports are given. Ballenger[5] has found the facial nerve involved in labyrinthine suppurations in 55 per cent of the cases under his observation, while the reports of Gerber[6] and Bezold[7] vary from 77 per cent to 83 per cent. Blake[8] states that the most vulnerable points of the facial nerve are the region above the oval window and the superior portion of the posterior tympanic wall.

A gradual progressive loss of hearing developing under close observation in a comparatively short time is characteristic of inner ear involvement. In diffuse infestive labyrinth-

itis of long standing there is generally found a total loss of hearing.

In the acute infective labyrinthitis, after three or four days, the acute symptoms disappear, but the nystagmus remains most marked to the sound side. There is now a condition known as the latent or chronic form of infective labyrinthitis. The nystagmus continues to the sound side for about three weeks and then becomes gradually weaker and finally disappears altogether. Headache is a common accompaniment of purulent labyrinthitis. The character is that of a dull, heavy pain referred to the temporal region on the side involved.

Tinnitus aurium occurs rarely in the course of a labyrinthine suppuration and only an occasional record is found among the many cases published.

Disturbances of equilibrium are fairly constant symptoms in the acute and subacute forms. In the chronic forms they are not so constant. Such disturbauces include giddiness, standing or walking, with closed or open eyes, and vertigo when even in a position of rest. The subjective vertigo complained of by the patient is a sensation of falling.

Kerrison[9] in a study of "Vertigo of Vestibular Origin" sums up his investigations by stating "There are two distinct forms of labyrinthine vertigo and associated ataxia,—one the familiar spontaneous type, due to vestibular irritation and occurring only during the acute stage of suppurative labyrinthitis; and the other not spontaneous, not constant, not necessarily accompanied by nystagmus, characteristic only of the lateral, or quiescent, stage of the disease, and induced solely by sudden and unexpected calls upon the lost or defective oriculation-sense, in the maintenance of which the intact vestibular organs are normally so important a factor."

An intratympanic examination in certain cases may reveal evidences of necrosis of the inner wall of the tympanic cavity, if pus is found exuding from the oral window or from a fistulous track through the promontory, a positive diagnosis could be made of labyrinthine suppuration.

The daily examination of the blood should be made in every case. In the acute form a high leucocytosis and increase in polymorphonuclear percentage are found. The large mononuclears are at times increased beyond their relative count.

Frequently when a case commences to subside, an examination of the blood will detect this condition several hours prior to the time the symptoms manifest themselves. In the chronic form there is little to be learned from the blood count before the operation.

DIAGNOSIS.

The diagnosis of labyrinthine suppuration can be made fairly accurate if in the presence of a negative caloric and total loss of hearing the other typical signs present themselves—such as a spontaneous nystagmus to the opposite side, nausea and vomiting may show along with a dull headache referred generally to the ear involved.

If in an acute suppuration of the labyrinth or in an acute exacerbation of a chronic purulent otitis media, the case should develop temperature, photophobia and headache in the presence of exaggerated reflexes, positive Kernig and positive chemical changes in the spinal fluid, a positive diagnosis of beginning meningeal involvement could be made and heroic operative measures undertaken at once. In the course of an acute focal infection, if there is evidence of an acute otitis media and the above symptoms appear, one is justified in performing the simple mastoid operation at once and exercising watchful waiting before draining the labyrinth and meninges.

In the chronic labyrinthine suppurations no definite symptoms may manifest themselves. Such cases are found in examinations of chronic suppurative otitis media cases where all the regular labyrinthine tests can be safely made. Even here the fistula test should be undertaken very carefully. From the clinical history much could be gained. It is among these cases that fistulæ are found occasionally leading directly into the labyrinth. I remember several cases studied at the University Hospital, Iowa City, with Dr. L. W. Dean, which presented well marked perforations leading directly through the promontory into the vestibule.

Impaired hearing, developing while the case is under observation and later having Weber's test localized in the sound ear, is generally evidence of invasion of the labyrinth. Accompanying this, the bone conduction is much impaired, and the range of audition greatly reduced, the upper tone limit

lowered and the lower tone limit elevated. Barany states that disturbance of equilibrium is characteristic only of the acute stage of labyrinthine diseases, and that it regularly disappears as the lesion advances either toward resolution or destruction of the vestibular structures. Von Stein,[16] however, holds that disturbances of equilibrium are present in all stages of suppurative labyrinthitis. When such disturbances of equilibrium are found in chronic suppurative labyrinthitis they are probably due to the overstimulation of the sound labyrinth. The caloric tests are negative. Rotary nystagmus to opposite side and the direction in which the patient would fall would always be influenced by the position of the head.

In a differential diagnosis, cerebellar abscess must be excluded. The nystagmus in a case of cerebellar abscess increases from day to day, while in suppurative labyrinthitis it becomes less and less marked and may finally disappear, owing to the extension of the suppurative process. In cerebellar abscess the quick component of the nystagmus is directed to the sound side and later it is directed to the diseased side. In labyrinthine suppuration the quick component may be directed for the first two or three days to the diseased side and then it is directed to the sound side. In cerebellar abscess the caloric tests are positive; no deafness present. Patient always tends to fall to the diseased side, irrespective of the position of the head. A vertical nystagmus is considered by some to be pathognomonic of cerebellar abscess. Choked disc may be present, whereas it seldom appears in labyrinthine suppuration. In some of those cases where the diagnosis before the mastoid has been opened is vague, they will become clear on opening into the tympanic cavity, and at this stage then and only then are some cases diagnosticated rightly.

Having excluded, by a differential diagnosis, other diseases, the diagnosis of chronic suppurative labyrinthitis can be made almost positive, when functional examination reveals total loss of hearing, negative caloric, Weber's test localized to opposite side, along with a shortened Schwabach and in addition the subjective signs of dizziness and spontaneous nystagmus. The diagnosis is confirmed when a fistula is found extending into a semicircular canal or any portion of the labyrinth.

In some few cases of localized purulent meningitis, the differential diagnosis may be difficult. A sudden rise in temperature, marked headache, vomiting, rigidity of neck muscles, positive findings in the chemical analysis of the spinal fluid, and in finding the diplococcus intracellularis meningitidis or pneumococcus in the spinal fluid points to meningeal infection.

Dench[11] states that in cases of beginning meningitis involving the vestibular nerve, where a nystagmus toward the sound side, indicating a paralysis of the affected labyrinth, may later diminish and disappear, and a nystagmus toward the affected side makes its appearance. This, he holds, is a positive evidence of a retrolabyrinthine lesion, which may be either in the cerebellar substance or in the meninges in the immediate vicinity of the vestibular nerve.

COMPLICATIONS.

The complications most liable to occur in suppurative labyrinthitis are meningitis, cerebellar abscess and septic thrombus of the jugular vein. Extension to the dura takes place through.

1. Perineural and perivascular sheaths of the seventh and eighth nerves.
2. The aqueductus vestibuli with consequent formation of an empyema of the saccus endolymphaticus and subsequent cerebellar abscess.
3. The aqueductus cochlea.
4. Erosion of superior and posterior semicircular canals.

PROGNOSIS.

It is difficult to state the prognosis of a labyrinthine suppuration. The acute form may subside gradually and healing take place. However, this may only be temporary, and sooner or later the condition returns. Every labyrinthine suppuration should be regarded as a dangerous process for the elimination of which we should use every possible means. We are not exactly able to distinguish between the favorable and the unfavorable forms. The prognosis is unfavorable in the chronic forms, perforation of the promontory wall and pressure of cholesteatoma extending into the interior of a semicircular canal.

According to Duel,[12] if the loss of function is sudden (within a few hours), following a virulent suppurative otitis media, or following a fistula test or any operative interference in the presence of perilabyrinthine inflammation, the case is probably an acute suppurative endolabyrinthitis. If such is the case there will be no return of function. On the other hand, if the function is lost slowly (days or weeks), even progressing to complete loss of both functions, there will be a final recovery or partial recovery of one or both, provided the labyrinth is not operated.

Politzer[3] holds that the prognosis is very unfavorable in an established perforation of the promontory wall, which, with a simultaneous deafness, must be looked upon as a sure sign of a diffuse labyrinthine suppuration and in which, as already mentioned, the destructive process not infrequently extends to the peripheral part of the internal meatus and to the auditory nerve.

An infection of the labyrinth of tubercular or exanthematous origin is generally held to be more destructive than due to other pyogenic causes.

TERMINATIONS.

According to Milligan and Wingrave,[13] the terminations of a labyrinthine suppuration may be as follows:

The acute diffuse pyolabyrinthitis may terminate in

1. Total destruction of the auditory function.
2. Sequestration of part or the whole of the labyrinth.
3. General blood infection.
4. Chronic disease.
5. Death.

The chronic pyolabyrinthitis may terminate in

1. Ossification of the affected area.
2. Caries of part or the whole of the labyrinth.
3. Erosion of bone and infection of the contents of the middle or posterior fossa.
4. Destruction of the static or acoustic segments of the internal ear.
5. Septicemia or pyemia.
6. Death.

TREATMENT.

There have been many different views held regarding the treatment of labyrinthine suppuration, and much confusion existed as to indications and contraindications of a labyrinthine operation. The treatment should only be considered after a most careful study of the existing conditions which are present in each individual case.

The purposes of the labyrinth operation, according to Ballenger,[14] are:

1. To prevent the extension of the purulent infection to the meninges and brain, and to check and cure an incipient meningitis having its origin in the labyrinth.

2. The second purpose is to cure an offensive purulent otorrhea which is perpetuated by a labyrinth fistula, remaining after the destruction of the labyrinth by a diffused suppurative labyrinthitis.

3. The third purpose is to relieve the patient of a distressing giddiness which occasionally persists after the cochlear nervous apparatus is destroyed by a suppurative process, while the vestibular nervous apparatus is but partially destroyed and gives rise to distressing and disabling giddiness.

The indications which are most generally accepted at the present time for opening the labyrinth are:

1. With the labyrinthine symptoms, complete deafness and nonirritability of the vestibular nerve of a chronic process or an acute exacerbation of a chronic process with beginning evidence of meningeal invasion indicate immediate opening of the labyrinth.

2. Necrosis of the labyrinth as induced by cholesteatomatous pieces discharging from the wound, and if associated with facial paralysis indicates opening of labyrinth and removal of all necrotic tissue.

3. Following a radical mastoid operation, at the first sign of meningeal irritation open and drain labyrinth.

4. According to Jansen,[15] any symptoms of cerebral complication makes a positive indication to at once open the labyrinth.

Generally speaking, there is no indication to open the labyrinth in an acute labyrinthine suppuration. However, the

case must be kept under most careful observation and at the first sign of meningeal irritation the complete labyrinth operation should be performed. In the acute tpye it is better to keep the patient at absolute rest. By that I mean no movement of the head or body. Ordinarily it will not require much will power to keep such a case at rest, for the patient will remain very quiet to avoid the most distressing symptoms that arise on movement. In certain cases, with marked onset and pronounced symptoms of the process becoming diffuse the vestibule must be opened, both in front and behind the facial ridge, the cochlea uncapped and the first and second turns opened so that the meningeal fluid washes through freely. A slit is made in the dura as near the internal auditory meatus as possible. The latent stage of labyrinthine suppuration is the period in which it can be operated upon with the best prognosis. Kerrison[16] states that vomiting, headache and elevation of temperature are distinctly characteristic of vestibular irritation and are not necessarily indicative of meningeal disease, and where these are not excessive or too prolonged surgical intervention should be delayed.

In cases of chronic middle ear suppuration, where there has been evidences of past suppurative labyrinthitis, and no meningeal symptoms present, it is best and safer to open and drain the labyrinth, at the same time performing the radical mastoid operation, and the granulations, if present, should not be curetted, as this would tend to break down the barrier to a point that would open a communication with the meninges through the aqueduct of the cochlea and vestibule.

According to such men as Hinsberg, Jansen, Neumann, Barany and Richards, it is most essential that the vestibule should always be opened and drained in the chronic labyrinthine suppuration, because

1. The operation secures free drainage from the vestibule, which is always involved in suppurative labyrinthitis, and

2. Enables the operator to destroy the membranous structure of the vestibular apparatus, which is a main point in contolling permanently vestibular symptoms.

The route posterior to the facial canal from the manipulative standpoint appears to be the most convenient, because of

least danger to the important structures, and allows the operator to fully expose the limits of the disease.

Much discussion has arisen during the past as to the nature and extent of the operation. Neumann[17] advocates the complete exposure of the cochlear and vestibular apparatus, while Alexander and others believe in only partial exposure. Operating during the latent stage of the disease, we are inclined to hold that a complete labyrinth operation should be performed if a fistula is found from which purulent secretion is discharging from the labyrinth or distinct evidence of necrosis is present in the vestibule or cochlea.

Ruttin has performed 100 complete labyrinth operations with one death resulting from the operation.

The primary step in every labyrinth operation is the complete exposure of the tympanic cavity and the horizontal semicircular canal by the radical mastoid operation. The internal wall of the tympanic cavity and the horizontal canal should be carefully examined for any fistulous tracks leading into the labyrinth. Should a fistulous opening be found in any of the canals they should be thoroughly opened and removal of all granulation tissue and necrotic bone. In many cases it will be necessary to chisel away the canals to their base, thus opening the vestibule above and behind. Where fistulæ are found, the labyrinth is better exposed behind the facial ridge. The posterior crus of the horizontal canal is opened and the anterior crus is chiseled away to the labyrinth. Then the bone lying below the oral window and above the round window is removed, thus exposing the vestibule and a small portion of the cochlea. The opening is now enlarged so as to freely drain the first and second turns of the cochlea. The posterior cranial fossa is not opened as a routine measure in the labyrinth operation.

Where meningeal symptoms are in evidence, then it is necessary to freely open the dura in the region of the internal auditory meatus.

If there is any evidence of remaining function in either the cochlear or vestibular apparatus, no operation upon labyrinth is justified in the absence of meningeal symptoms. The opinions differ as to the advisability of opening a dead labyrinth while performing a radical mastoid. From the recent lit-

erature and the opinions of our most experienced men, we learn that in the presence of a dead labyrinth the radical mastoid operation should only be performed unless there is direct evidence of meningeal irritation previous to the time of operating. It is pointed out elsewhere in this paper that the danger of opening a dead labyrinth in the absence of meningeal symptoms would likely convert a latent focus into an active affair which, by breaking down the barriers nature had so well established, would tend to create a beginning meningitis.

The method of Hinsberg, Bourguet and Botey are very much similar. They all reach the vestibule from in front of the facial ridge and then open and drain the promontory, etc.

Many operations have been advocated for purulent labyrinthitis, but the truth is made even more evident that we must make the surgical technic meet the desired condition in each individual case.

TWO CASES REPORTED BRIEFLY.

Case 1.—In August, 1916, Miss K., age 42, gave a history of chronic discharge from the left ear for the last seventeen years. During the last three months she has noticed peculiar dizzy spells, which forced her to bed at different times. The discharge had become more offensive, and with the advent of the dizziness had compelled her to seek relief.

Examination showed the canal full of thick purulent material, culture from which revealed a mixed infection, with the streptococcus predominating. There was a negative caloric and patient could hear loud spoken voice at two feet with Barany's apparatus in the right ear. There was a spontaneous nystagmus to the right, and Weber's test was also localized to the right ear. Temperature, 99.4; Wassermann negative, as was also general examination. No fistula or turning tests tried. Blood showed whites 9,900, polys 78 per cent.

Diagnosis: Otorrhea chronica left with acute involvement of labyrinth.

Operation.—A radical mastoid was performed, exenterating all the necrotic cells. Leading directly into the external horizontal canal was a well marked fistula, from which a seropurulent material was oozing. As there was good drainage

and no symptoms of meningeal irritation it was decided not to open the vestibule or cochlea until such time as called for.

Following the operation there was a profuse drainage through the fistula from the labyrinth. This gradually diminished in quantity, becoming more fluid like. There continued an uneventful recovery. Patient was advised of the necessity of frequent observation because of the labyrinthine involvement. The spontaneous nystagmus gradually lessened and finally disappeared six weeks after the operation. Six months later the patient appeared. There was a slight moisture in the tympanic cavity, but no evidence of any further necrotic areas present. There was no dizziness, and the patient had gained some fifteen or twenty pounds in weight.

Case 2.—In March, 1918, Mr. R., age 32, farmer, came for relief of chronic suppuration in his left ear, which had troubled him for the last eleven years. At times the discharge would apparently cease and then, following a cold, it would bother him again.

Two months ago, following an attack of measles, the discharge became very thick, and he noticed for the first time that he was becoming dizzy. These attacks became more frequent and lasted longer intervals until he was barely able to be up only part of the day. On several occasions he had become very nauseated and vomiting had followed.

Examination revealed a fairly well nourished male, whose chief complaint was the chronic discharge and dizziness of the left ear. The canal was full of foul smelling pus, caloric reactions negative and absolute deafness was present. The general examination was negative. The patient was sent to the hospital to be prepared for the surgical work, and during the night his temperature suddenly rose to 103, patient unable to sleep and a marked headache was present. The fundus examination showed a congested disc and spinal puncture revealed the fluid under pressure with 2,000 cells per cubic millimeter and globulin test positive.

A diagnosis of beginning intracranial irritation was made and immediately a radical mastoid was done, and the vestibule opened, both in front and behind the facial ridge. The first and second turn of the cochlea was also uncapped. There was no evidence of any perforations in the promontory, nor

were there any fistulæ found. There was a slit made in the dura close to the internal auditory meatus. For the next four days the dressings were saturated with cerebrospinal fluid. There was a spontaneous nystagmus to the right side. The patient gradually cleared up, his temperature and blood pictures gradually coming down to normal. The spontaneous nystagmus lasted four weeks. Patient, while in bed, complained of objects rotating about him. However, this phenomenon gradually cleared, and patient was discharged from the hospital some seven weeks following the operation.

Three months following this the patient reported and he was feeling much better, had gained some weight, but there was still some dizziness left.

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XXXV.

THE APPLICATION OF DENTAL MOLDING COMPOUND FOR THE MAINTENANCE OF SKIN GRAFTS IN THE MIDDLE EAR AND MASTOID CAVITIES.

By Joseph Israel, M. D.,

Houston, Texas.

The purpose of the radical mastoid operation is known to us all, but the question arises—do we achieve the desired results, which are: the union of the external auditory canal, the middle ear, antrum and mastoid wound, into a common cavity and the lining of this cavity with epidermis through the implantation of a flap from the external canal.

The necessity for skin grafting in the middle ear and mastoid cavity as a secondary procedure, following the radical mastoid operation, is apparent.

The plastic flap, used by a large majority of otologists, works well in some cases. In the author's experience in over 100 radical mastoid operations, epidermization of the cavity, by the flap method is not always obtained. It is then that the necessity for the use of the detached graft is manifested.

The author is skeptical as to the use of the pedunculated graft of the flap operation, because pedunculated flaps must be thick. The attachment of the flap in place by sutures throws tension on the pedicle. Passive congestion may ensue, which results in death to all parts.

In dermic and epidermic detached grafts the life of the graft depends upon its contact with the underlying surface. If thorough contact is obtained and the proper surgical technic is carried out, the dangers of the graft not taking are remote. The question arises as to the securing of perfect approximation of these two surfaces. It is at this point I wish to call attention to the use of the dental molding compound.

I have used this substance in two types of cases:

First—In those cases where complete obliteration of the mastoid and middle ear cavities is desired, and,

Second—In cases in which there is a persistent otorrhea, due to infection in the eustachian tube, and when it is desired to obliterate the middle ear orifice of this tube.

In the first instance, when it is desired to completely obliterate the mastoid and middle ear cavities, after having the wound prepared for the reception of a skin graft, a sheet of sterile dental molding compound is immersed in hot water for a period of thirty seconds, when it is taken out and allowed to cool, so that it remains flexible though not gummy. The compound is then packed into the mastoid cavity and middle ear and allowed to remain until it hardens, when it is removed.

With this impression of the cavity, the graft is then taken, preferably from the thigh, taking enough epidermis to cover the cavities. It is then spread over the impression compound, raw surface outward, seeing that there is no turning in of the edges. This done, the molding compound, with the graft lying over it, is inserted into the mastoid wound and firm pressure is made, so that it will slip into place and approximation of the surfaces obtained.

The mastoid wound is allowed to remain open. A gauze pad is placed over it. Drainage is obtained through the external auditory canal by a gauze wick, and a pressure bandage is applied to the mastoid wound. The bandage is removed in four days, and the pressure compound taken out, and the wound is closed under local anesthesia.

The second variety of cases, as before stated, are those where the disease is confined to the eustachian tube, and after thorough curettage it is desired to obliterate the orifice of the tube by skin graft.

For this type of case, the writer introduces the impression compound through the external canal into the middle ear cavity, taking an impression of the part of the cavity that it is desired to obliterate, namely, the eustachian orifice. A skin graft is taken and placed over the impression compound, raw surface outward. It is then gently introduced through the external canal into the middle ear cavity. Firm pressure is made, so that approximation of surfaces is obtained. The

compound is held in place by cotton packs, together with a pressure bandage over the external canal.

After four days the bandage is cut, and the impression compound gently drawn out of the external canal with a pair of forceps.

CONCLUSION.

The success of a skin graft, in no matter what part of the body, depends upon approximation of the surfaces. This can only be brought about by pressure. It is for this reason that the author has used this method of procedure.

403-407 CARTER BUILDING.

XXXVI.

SOME REMARKS ON THE INDICATIONS FOR SIMPLE MASTOID OPERATION IN THE ADULT.

By John B. Rae, M. B., C. M.,

New York.

We arrive at a diagnosis of mastoiditis on information which we receive from three sources: (1) The general or constitutional symptoms; (2) the information which we receive from the ear itself; (3) the laboratory aids.

The temperature in uncomplicated mastoiditis is low and may be normal, even in the worst cases. There is general malaise, loss of appetite and coated tongue. Pain may or may not be an evident symptom. When present it is referred to the temporal region, is dull and aching in character, is worse at night and sometimes disturbs or prevents sleep. Pain may also be entirely absent.

On inspection of the canal and drum head, we will find an acute or chronic otitis media, or there will be evidence or history of a recent involvement of the middle ear. It is generally conceded that a purulent middle ear always precedes a pus mastoid, even although the history may be indefinite and the local appearances little suggestive. The location and size of the perforation in the drum head, should that be present, will be noted, and its adequacy or inadequacy for drainage will be considered. An effort should be made to estimate the amount of discharge. This is done by wiping out the canal. The patient is then directed to Valsalva, and the amount of pus forced through the opening into the canal should be noted. This procedure may be repeated until the middle ear cavity is emptied, and in this way an estimation of the amount of discharge may be arrived at, at one sitting. This estimation of the amount of discharge is important, as in the absence of other symptoms this may be the deciding factor in determining operation. Acute suppression of discharge with increase of pain and temperature is also quite significant. Presence or absence of prolapse of the posterosuperior canal wall

should be looked for. This is an edema at the junction of canal wall and drum head. The acute fold at the junction is lost, and it is impossible to say where the canal wall terminates and drum head begins. It is most likely due to swelling of the periosteum caused by involvement of cells in the neighborhood of the posterior canal wall or of the canal wall itself.

Swelling behind the ear is a late symptom of mastoiditis in the adult. This swelling may be simple edema or subperiosteal abscess. Needless to say, in either case this would be an indication for operation in the adult. Swelling behind the ear is not always due to mastoiditis and will call for a differential diagnosis between that due to mastoiditis, to furunculosis and to local cellulitis.

When slight in amount postauricular edema is detected by standing behind the patient and observing both ears at the same time. Obliteration of the postauricular fold can be thus observed, even though displacement of the auricle is scarcely perceptible otherwise.

Perhaps the most constant symptom of mastoiditis is tenderness on pressure. This is elicited by firm pressure inwards and backwards over various points of the mastoid process. Care must be taken that no movement of the cartilage is made, as this will cause great pain if we have a furuncle in the canal. Tenderness on pressure is due to direct compression of the inflamed bone and not to periostitis, which is a late modification. Tenderness is as a rule an early symptom. The extent of the tenderness is mainly determined by the character of the mastoid. It is elicited over the antrum, over the tip and over the whole surface of the mastoid if this is very cellular and involved. It is a mistake to speak of tenderness over the mastoid emissary vein, because we can never be certain of the location of this vessel. Tenderness on pressure may be entirely absent. This may be due to an excessively thick cortex, to a very forward sinus, leaving little space for cells, or to sclerosis of the entire mastoid process.

The laboratory aids have references to the identification of the infecting organism, to the total and differential blood count, to the blood culture and to the X-ray findings.

While it may be of some value to know the causative organism, too much stress should not be laid on this. Clinical

observation of the progress of the disease is much more important. If we presume, for example, that a staphylococcus infection is necessarily a mild one, we may adopt an attitude with regard to a particular case that is not at all justified by its clinical course. It is the experience of most of us that any infecting organism may in any individual case give rise to the greatest destruction. It is therefore wise to emphasize the importance of clinical observation while by no means minimizing the value of the laboratory aids.

The blood counts and culture are not of any great value in uncomplicated mastoid cases. It is only when the deeper structures are involved or threatened that they are really called for.

It is equally true that there is usually no need for X-ray examination in the average case of acute mastoiditis. Apart from the information which the picture gives to the observer, the exhibition of the X-ray plate to the patient or his friend may have considerable influence in procuring permission for operation.

In the X-ray examinations there should be the closest cooperation between the otologist and the radiologist. The latter should receive from the otologist sufficient information about the patient to give him an intelligent idea of the clinical situation. The picture having been taken, both observers should make a record of their interpretations and these should always be checked up in cases which come to operation. In this way a just estimate of the value of this aid to diagnosis will be reached and both clinician and technician enabled to interpret the pictures to the advantage of the patient. We have not yet arrived at the point at which pictures can always be absolutely interpreted, and too often do we find that the report is ambiguous and undecided in those very cases in which we had hoped that the X-ray might prove to be a decided factor and enable us to set a definite course.

With all or a majority of these many symptoms present, the diagnosis of mastoiditis is not difficult, nor can the indication for operation be missed when they progress or even continue without increase, over a period of a few days. If asked to state in a word the indication for operation, the writer would answer that operation is indicated as soon as we are

assured of the presence of pus in the mastoid. He can see no reason for delay once that conclusion has been reached.

It must be remembered that in a number of cases in which we have to deal with a mastoiditis complicating an acute middle ear, a distinct improvement occurs in all the symptoms when a myringotomy has been performed and treatment started with the patient at rest in bed. Pain subsides, temperature approaches or reaches normal, and the mastoid operation may have to be recommended in spite of this apparent improvement.

We must also be on our guard against the natural tendency of a patient to minimize or hide tenderness on pressure. This applies particularly to observation cases in hospital who have had the chance to see other patients returned from the operating room and to witness dressings. They are anxious to avoid operation and will sometimes do their utmost to delude themselves and deceive the examiner.

In addition there can be no doubt that in unoperated cases of mastoiditis the patient may rapidly acquire a complete tolerance. It is not an uncommon experience to find a patient who has been attending to his every day affairs over a period of weeks without especially complaining of his mastoid, which has been slowly going from bad to worse.

It is in the cases of quiet mastoiditis in which most of the classical sympotms are lacking, that the judgment, firmness and courage of the otologist will be called for. With the temperature normal and the patient free from pain, the operative indications may be very limited in number, and apparent and urgent only to the otologist and not at all so to the patient and his friends. Needless to say, the quiet case is particularly urgent, because of the danger of the onset of complications during a period of procrastination, which to the unskilled observer seems absolutely justifiable. The X-ray here offers its best field of usefulness, and if the findings are positive immediate operation should be urged.

The clinical evidence in these quiet cases has to do with the appearance in the canal, the amount and character of the discharge, the presence of persistent tenderness on pressure or, less frequently, the persistence of an otherwise unaccountable temperature. It is also quite noteworthy that the tongue often

remains coated. With regard to the canal we find marked prolapse of the posterosuperior canal wall or more frequently a progressive narrowing of the entire fundus. The visible drum area is quite small, and if discharge be present it may show as a small pulsating bead at the inadequate opening in the drum membrane. Parenthetically it may be stated that these are the cases in which repeated myringotomies are sometimes performed. It is a safe general statement that when one or two myringotomies, made under favorable conditions, fail to secure adequate drainage and bring about relief, that opening of the antrum is a really conservative measure and that this is indicated rather than further myringotomies.

In those cases in which excessive amount of discharge is the characteristic symptom, the pus may be thick and creamy and adhering to all the canal walls so that it fills the lumen of the speculum when introduced. When wiped out the canal will fill up again immediately, when the patient is directed to valsalva. We conclude that this amount of pus is not coming from the middle ear alone.

Tenderness on pressure when present does not differ in location or degree from that described a few minutes ago, and depends on the character of the mastoid itself and the extent of involvement.

We must not forget that in the quiet type being described the decision for or against operation may have to be made not in the presence of all of these groups of symptoms but in the presence of only one. And also that the general condition and feeling of the patient are such that the gravity of the situation is by no means apparent to the patient himself, to his friends or even to his general medical adviser.

In conclusion, then, in the average case in which all the classical symptoms continue or increase for a few days after myringotomy and treatment, we may be reasonably certain of the presence of pus in the mastoid and advise operation.

It is the opinion of the writer that no definite limit in days should be set down as a safe observation period, but that each case should be considered on its merits, particularly from the strictly clinical standpoint, and with regard to increasing or decreasing severity of the symptoms and limitation or extension of the pathologic process in the bone. The average case

allows plenty of time, say up to a week, for observation in the absence of any urgent call, and experience proves that cases so held over do better after operation when nature has had a chance to collect the constitutional and local resources in the fight against the disease.

In the quiet cases an X-ray is immediately called for. Should this show bone destruction or be otherwise positive, immediate operation is indicated. The contrast between the healthy and the diseased side may prove to the patient that his condition is more serious than he had imagined. It is a constant source of astonishment to find the amount of destruction that has taken place in these cases with a minimum of symptoms.

Should the X-ray be indefinite, operation should still be urged.

It is often observed in these doubtful cases that the hearing is diminished out of proportion to the apparent lesion, and this is of considerable diagnostic value.

It seems to the writer that the correct attitude of the otologist in face of a quiet mastoid should be to ignore the negatives, to dismiss from his mind what is lacking in the symptomatology and to concentrate on what is positive, even should that be but one lone symptom.

The writer has yet to regret opening a single quiet mastoid in which the average observer has made the clinical diagnosis.

XXXVII.

SPECIMENS ILLUSTRATING OPERATIVE AURAL SURGERY AND THE MAKING BY GRADUATE STUDENTS OF PREPARATIONS SHOWING AURAL SURGICAL ANATOMY.

By E. B. Gleason, M. D.,

Philadelphia.

The writing of this paper was inspired by the statement of George E. Shambaugh in his introduction to the chapter on the surgical anatomy of the ear in Loeb's "Operative Surgery of the Nose, Throat and Ear." "The first problem for the surgeon who would undertake the operations on the ear is to master the details of the anatomy of this region. This cannot be acquired from textbooks, nor is this knowledge readily gained by attempts to do these operations on the cadaver. A thorough grasp of the complicated anatomy of the temporal bone is best acquired by the study of preparations made especially to show this or that relation. The knowledge comes through the actual making and handling of such preparations."

As to preparations to be actually handled by students in illustrating a cadaver course on the surgery of the ear, a minimum outfit should consist of specimens showing each of the more common operations, either on macerated and bleached skulls, dried heads or wet preparations, supplemented by anatomic preparations on detached temporal bones showing the more common anomalies. Briefly it is as follows:

1. The temporal bone of an infant at birth, separated into the annulus, ossicles, squamous and petrous portions.

2. The simple and radical operation on the skull of an infant at birth.

Supplementary specimens. 2A. Malleus, incus, stapes; 2B. Malleoincudal articulation on the "scute." 2C. Ankylosis of this articulation.

These specimens show the ossicles, tympanum, antrum and internal ear of practically the same size as in the adult, devel-

opmental changes being due to the growth of the mastoid process with the formation of pneumatic cells, should these form; and to the growth of the external auditory canal (tympanic bone), resulting in a changed position of the stylomastoid foramen. It is no longer on the external surface of the skull as in the infant. Consequently, the lower portion of the primary incision of the mastoid operation should be further back than in an adult. The antrum is somewhat posterior to the posterior tubercle of the annulus and between it and the linea temporalis.

Preparations illustrating ear operations are better shown upon a half head or whole skull, from which the calvaria has been removed, than on detached temporal bones, because the average student has difficulty in visualizing the relationship of a detached temporal bone to the skull. He usually holds a temporal bone as if the squama and inner wall of the tympanum were vertical and the facial canal sloped downward, backward and outward from it, while in the skull the descending portion of the facial canal is always vertical and the plane of the inner wall of the tympanum is downward, inward and forward. Hence the antrum is always more superficial than the rest of the tympanum.

The specimens should be of such a character as not to be readily injured by rough handling or contact with dirt. They are supposed to be handed around while students are at work and occasionally dropped on the floor and otherwise maltreated.

LANDMARKS FOR THE MASTOID OPERATIONS.

3. In this skull, a portion of the descending part of the facial canal is laid open and tinted red; the contour of the surrounding parts has been preserved as much as possible, particular pains being taken to preserve the pyramid that contained the stapedius muscle in order to show what portion of the inner wall of the canal can be removed in the radical mastoid operation without endangering the facial nerve. The posterior portion of the prominence of the horizontal semicircular canal has been uncapped in the position where it is most frequently laid open by a careless operator in mastoid operations. On the other side of the specimen the superficial landmarks for the

mastoid operations are shown, and the semicircular canals laid open and the tegmen removed. The line of the descending portion of the facial nerve is a straight vertical line between the stylomastoid foramen and a point just posterior to the oval window. The root of the styloid process can usually be felt with the finger tip during a mastoid operation. Under such circumstances, the stylomastoid foramen lies immediately beneath the finger tip, and if in a radical mastoid operation, when the stapes can be seen, the line of the facial nerve is a straight line between the finger tip and a point immediately posterior to the stapes. A very important part of a cadaver course is the exposure of the horizontal and vertical part of the facial curve. The cleavage planes of the bone are such that the vertical portion can be laid bare by two strokes of the chisel so directed as to remove a triangular piece of bone between the oval window and the stylomastoid foramen; but by a student the bone is much better removed layer by layer; the curve of the nerve downward behind the oval window where the bone over the nerve is very thin being the starting point and principal landmark for the dissection.

Supplementary Specimens.— 3A. Superficial landmarks. 3B. Section parallel to the meatus in a small celled pneumatic mastoid. The tegmen has been removed to show the position of the "boss" of the external semicircular canal, the ossicles and other "internal" landmarks.

3C. Radical mastoid with horizontal and vertical portions of the facial nerve laid open. Both the external and the posterior semicircular canals are laid open. Part of the tegmen has been removed and the dura exposed. Nearly all the accidents that could happen to an unfortunate operator in a mastoid operation are shown in the specimen.

3D. This specimen shows an extremely superficial and anterior position of the sigmoid sinus, so that it would be difficult to expose the antrum by the usual method without wounding the sinus. The bone is of the infantile type and the antrum very small. (See also 4A and 4B.)

THE MASTOID OPERATIONS.

4. A skull on one side of which a simple mastoid operation has been done and on the other a radical. A comparison of

this specimen with the dissection of the facial nerve in No. 3 shows how much of the wall between the tympanum and the artificial cavity in the bone it is safe to remove in a radical mastoid operation. At the inner portion it is rather greater than would appear safe to the uninitiated.

Supplementary Specimens.—4A. The Stacke's operation. From this operation, by combination with the simple mastoid operation, was developed the modern radical mastoid operation. A modification of Stacke's operation is occasionally performed at the present time in connection with the removal of the ossicles in chronic otorrhea.

4B and 4C. These specimens illustrate the difficulty of exposing the antrum when the sigmoid sinus is superficial and far forward. In both the specimens the antrum is of normal size. However, the difficulty of reaching the antrum is not as great as in specimen 3D.

THE SIGMOID SINUS, OTIC BRAIN ABSCESS AND INTERNAL EAR.

5. One side of this skull shows the operation for exposing and opening the sigmoid portion of the lateral sinus. The operation has been done on a dried head, the dura remaining intact. The other side of the head shows a Jansen-Neumann operation and a detached temporal bone S. A., the simpler Hinsburg operation. Both specimens show the removal of the tegmen and the removal of the bone of Troutman's triangle, with exposure of the dura as in the search for a suspected brain abscess in these localities.

Supplementary Specimens.—5A. 1. Temporosphenoidal abscess. 2. Jansen-Neumann operation without exposing the dura. 3. Removal of bone for temporosphenoidal abscess. 4. Perisinus abscess. 5B. Hinsburg operation.

THE INTERNAL EAR.

6. A skull with the semicircular canals of both ears laid open.

Supplementary specimens:

6A. Specimen showing the proximity of the internal carotid to the anterior portion of the first turn of the cochlea and the eustachian tube.

6B. Topography of the horizontal portion of the facial nerve in relation to the vestibule and cochlea.

6C. Vertical portion of the facial nerve.

6D. The vestibule, showing the position of the ampulla and indifferent ends of the semicircular canals.

PREPARATION OF SPECIMENS SHOWING THE SURGICAL ANATOMY OF THE EAR BY GRADUATE STUDENTS.

As in dentistry a candidate for the degree of D. D. S. is required to present a specimen or his skill in making artificial dentures, so a graduate student should be required to present specimens of his work showing the surgical anatomy of the ear. In the dead room a work bench, motor, burrs, and other tools and instruments necessary for the purpose should be at his disposal.

He should be encouraged to produce specimens rivaling in beautiful display of anatomic details those imported from France, but at present nothing more is contemplated than the production of specimens showing the structures of most surgical importance in the more common operations upon the ear.

7. This specimen is especially recommended as an example of this type. The anterior wall of the external auditory canal has been removed to show the external surface of the drum head. The posterior portion of the internal meatus has been removed to afford a view of its fundus. The tegmen has been removed and shows the antrum with the cellular structure of the bone surrounding the antrum The inner surface of the drum head, ossicles and muscles are plainly in view. Trautman's triangle and the capsule of the labyrinth are shown. The capsule is laid open in such a manner as to show the vestibule, semicircular canals, cochlea, the first part of the course of the facial nerve within the temporal bone and the geniculate ganglion. The surgical relations of the sigmoid, inferior and superior petrosal sinuses and the bulb of the jugular vein are shown. A sufficient amount of the upper portion of the carotid canal is removed to show the proximity of the internal carotid to the cochlea and eustachian tube.

8. Is a similar dissection of the macerated and bleached temporal bone.

9. Is a dissection made in such a manner as to show better than specimens 7 and 8 the course of the facial nerve and the relation of the cochlea, vestibule, horizontal and superior semicircular canals to the inner wall of the tympanum. All of these structures are laid open but the fenestra remain intact. It is possible for a student to make either of the specimens 7, 8, 9 in two or three hours; but a longer time is well spent, as a familiarity with the important anatomic structures of the temporal bone and their relationship to each other is best acquired by the slow and careful making of such specimens. It is probable that specimens made with a burr are superior to sections made with a saw for learning the anatomy of the parts. However the methods can sometimes be combined to advantage, as in 9 and 10. The most useful sections for displaying important anatomic structures are vertical and horizontal sections through the external and internal auditory meati; and vertical sections through the tubotympanic axis and through the petrous bone parallel to the tubotympanic axis.

10. Is a section through the tubotympanic axis enabling the worker to readily open the labyrinth from the inner wall of the tympanum. Trautman's triangle has been partly removed in order to better show the capsule of the labyrinth. This is laid open. The fenestra are left intact. The horizontal and vertical portions of the facial canal are better shown in this specimen than in most of the others.

Dr. Frank A. Bridgett has kindly loaned for inspection some of the specimens he made during his cadaver course a year ago.

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of December 5, 1921.

The President, Dr. Robert Sonnenschein, Presiding.

Epithelioma of the Tongue.

Dr. Alfred Lewy showed a patient who had first been seen by him in the latter part of September, 1920. He had complained for two months of discomfort, rather than actual pain, at the right side of the root of the tongue, had lost some weight and did not feel quite as well as usual. Inspection showed three raspberry-like prominences, with an irregular fissure, situated at the right side and dorsum of the tongue, adjacent to the right tonsil, and some infiltration of the right posterior pillar. Examination for tuberculosis and syphilis being negative, he proceeded on October 9th to coagulate the entire area, using the D'Arsonal current, about 1200 m. a. The slough was separated in about two weeks, the patient suffering more or less pain in the meantime. Histologic section of tissue, made at the time of operation, was exhibited and showed the epithelium invading the underlying tissue at one point.

Several weeks after the operation the gland bearing tissue of the right side of the neck was removed. Examination of this glandular tissue revealed no regional extension of the epithelioma. The patient was subsequently X-rayed. Following this the operated region in the side of the neck became hard and swollen, but this had since subsided to a slight infiltration.

Several rough teeth had been extracted, and the patient wore a little hard rubber prothesis to protect the edge of the tongue from friction. There appeared to be a mild grade of parasthesia of the tip of the tongue.

He had gained in weight and felt very well. His general appearance was excellent. He had been taking intermittently a colloidal preparation of arsenic.

Dr. Lewy stated that in another case there was a carcinoma of the tonsil, which he operated by the same method as that used in the case he showed. That patient remained well for nine months and died of metastases. There was no recurrence in situ, and in that case the pain was relieved immediately. There was a large slough in the throat, which separated without trouble. The other two cases went on from bad to worse without any benefit whatever.

Bilateral Labyrinthitis Following Gonorrhea.

DR. GEORGE W. BOOT presented a patient with bilateral labyrinthitis following gonorrhea. This patient, a white man of about forty, had had a chronic gonorrheal urethritis and prostatitis for about a year. On August 10, 1921, while under treatment for the urethritis he complained of deafness. On October 8th, he complained of head noises. On October 13th he was unable to hear any of the tuning forks by either air or bone conduction. On that date there was no spontaneous past pointing. He had a marked swelling in the lumbar region, which was red and angry looking, and the whole region was much infiltrated. X-ray examination of the back was not satisfactory. On November 6th, deep fluctuation of the mass in the lumbar region was detected, but attempts at aspiration were unsuccessful. Rotation tests on November 8th gave the following: No spontaneous past pointing; after rotation to the right, momentary nystagmus only; no past pointing; after rotation to the left, momentary nystagmus only; momentary past pointing.

Diagnosis: Chronic gonorrheal urethritis; osteoarthritis of the lumbar spine; total labyrinthine deafness on both sides with total loss of vestibular function.

The Wassermann reaction was negative. A small swelling on the forehead which suggested gumma proved on excision to be a small lipoma. In spite of the total loss of function of both vestibular systems, the patient had never been markedly dizzy and had never been unable to walk. The symptoms connected with the eighth nerve ran their course in about a month.

Whether the rapid labyrinthitis was due to a gonorrheal infection of the labyrinths, Dr. Boot was unable to say, but thought it seemed quite as likely as anything else, and in view

of the known occurrence of gonorrheal arthritis, gonorrheal iritis and gonorrheal spurs on the heel, it seemed to him more likely than that the labyrinthitis should be due to an accompanying infection by other pus organisms.

Dr. Boot also presented a patient with a tumor of the left vagus. This was a colored man, forty years of age. His illness began about February 15, 1917, as a sensation of pressure about the neck and upper part of the trunk. This was followed by a dry strangling cough, difficult breathing and often blindness and unconsciousness. The unconscious spells were accompanied or preceded by convulsions and had occurred as often as ten times an hour. The spells were particularly apt to occur with the approach of sleep, and the patient felt as if he were choking to death. On admission to Cook County Hospital his complaint was of pain on left side of neck and cramps of the muscles of the left side of neck.

He had malaria in 1908, gonorrhea in 1911 and an aural discharge in 1901. He had been married sixteen years and has two children alive and well. There had been no deaths in his family. He had vomited occasionally and had lost twenty-five pounds in the last six months. He has a cough before the onset of a convulsion. Examination of the neck showed a small tumor mass about 1 cm. in diameter lying just anterior to the sternomastoid muscle, opposite the upper part of the larynx. This tumor was firm, movable and apparently lay inside the carotid sheath. Pressure on it caused pain and cough. If firm pressure was continued the pulse slowed markedly and on auscultation the heart sounds disappeared. A convulsion then appeared. Examination of the larynx showed the left vocal cord to be paralyzed in adduction. This patient had a fibroid tumor of the left vagus with abductor paralysis of the larynx and the Stokes-Adams syndrome. The Stokes-Adams syndrome could be produced by pressure at any time. (The accompanying pulse tracing shows how pressure on the tumor caused slowing of the pulse.)

DISCUSSION.

Dr. J. Holinger was interested in the first case because of the finding of labyrinthitis of gonorrheal nature. Labyrinthitis from gonorrhea was never heard of. In his opinion, a

labyrinthitis would have much more stormy symptoms than those present in this case. He believed the complement fixation test would show whether there were toxins or an acoustic neuritis. He thought the tumor on the forehead was a gumma. It looked and felt like a gumma and was in the typical position. The fact that the Wassermann reaction was negative proved nothing and he advised the use of injections of arsphenamin. He believed the case had nothing to do with gonorrhea but was a typical syphilitic deafness.

Dr. Alfred Lewy thought Dr. Holinger's explanation was the feasible one. They had not excluded syphilis. It might also be possible for bony changes at the internal auditory canals to shut off both nerves.

"The Efficiency of Artificial Aids to Hearing."

Dr. Paul E. Sabine, Riverbank Laboratories, Geneva, Illinois (by invitation), presented a paper on "The Efficiency of Artificial Aids to Hearing."

ABSTRACT.

This paper gave the results of tests made upon eleven different devices for the aid of the deaf, made at the Wallace Clement Sabine Laboratory, Riverbank, Geneva, Illinois. The method employed was a modification of that developed by Professor Wallace C. Sabine for the study of problems in architectural acoustics.* The observations were made by a deaf observer, the cause of deafness, as diagnosed by Dr. J. Gordon Wilson, being fixation of the stapes with no involvement of the inner ear. Sound from a set of calibrated organ pipes was produced in a large empty room. The time required for the sound, as it died away after the pipe had ceased to speak, to fall to the threshold of audibility was determined. Under the same conditions this time was measured when the particular device being tested was applied to the ear of the observer. From the difference of these times, the amplification produced by the instrument was computed. The experiments covered the range of tones from 128 to 4096 double vibrations.

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*See Proc. of the American Academy of Science, Vol. XLII, No. 2, June, 1906.

Lantern slides were shown giving the amplification for the various tones tried. For instruments of the open trumpet type these amplifications were found to follow the order of size of the instruments. The effect of the natural tone of the air column enclosed by the walls of the trumpet were shown in increased amplification of these tones. The maximum amplification produced by the largest instrument tried was about twenty fold. Instruments of the trumpet type, modified with the view of reflecting and focusing the sound, gave no greater magnifications than open trumpets of the same size. The method employed in these tests did not permit of conclusions being drawn as to the efficiency of speaking tubes.

Instruments in which the vibrations of diaphragms held in contact with the teeth and thence, by bone conduction, are carried to the auditory nerve, were found to produce positive, though slight amplifications for low pitched sounds, but were ineffective in the case of tones above middle C.

Telephone devices produced relatively large amplifications and showed a marked increase in efficiency for tones near the natural frequency of vibration of the transmitter and receiver diaphragms. Thus one device gave a magnification of three hundred fold for tones in the neighborhood of 100 vibrations per second, which was the natural frequency of the transmitter diaphragm.

By comparison with the duration of sound audible to normal ears under identical conditions, the amplification necessary to give the ear of the deaf observer a sensitivity equal to that of normal hearing was computed. This varied with the different tones but was of the order of 100,000.

It was pointed out that the discrepancy between the amplification produced by the best of the hearing devices and that required to produce normal sensitivity does not argue the entire inadequacy of hearing aids. Measurements show that sounds of ordinary loudness, such as speech sounds, for example, are of the order of 1,000,000 times the threshold intensity. Hence a hearing device which produces an amplification of 300 fold without distorting the quality of the sound and without the extraneous sounds occurring in telephonic devices would prove an extremely valuable aid in any but extreme cases of deafness, even though the deaf ear thus

aided would be much less sensitive to faint sounds than are normal ears.

Dr. J. Gordon Wilson and Dr. John P. Minton presented a paper entitled:

"The Minimum Audibility of Hearing in Normal and Defective Ears."

ABSTRACT.

"Only within the last few years has there been any marked attempt to study deficiency of hearing by the use of precise physical measurements carried on by a physicist in cooperation with an otologist. The work which the writers have been carrying on during the past two years is an example of such a cooperation, which it is hoped will be productive of an increase in our knowledge, physical and physiologic, of audition. The correlation of data, taken by a physicist and by an otologist, promises to yield results of fundamental importance and of practical value. This work has been made possible through the development during recent years of the vacuum tubes and through the increased knowledge of telephone receivers.

"Experimental Method.—The audion oscillator, a telephone receiver tuned to a natural period above the range over which the tests are being made, and a Wheatstone bridge circuit are employed to make measurements of the current which makes audible tonal vibrations in the telephone receiver. The electrical and the vibrational characteristics of the receivers are determined so that the vibrational energy of the diaphragm can be calculated for ears with normal or defective hearing. Curves are then plotted showing the reciprocal of the minimum audible energy at the different pitches up to 5,000 d. v. Curves are also plotted for ears with deficient hearing showing at the deficient pitches how much more current through the receiver is required for the patients to hear than is required by a normal ear; such curves show the relative minimum audible current at the various pitches compared with the normal.

"Experimental Results.—Tests which have been made on normal ears—that is, ears which when examined by an otologist show no physiologic defect, show that the maxima of sensitivity occur at various pitches, but these maxima are not in the same position for all ears. There is also a wide difference in sensitivity among normal ears, but in general they

are all alike in that they are most sensitive from 300 d. v. to 5,000 d. v., the range in pitch which covers the regions most important for speech and music. By precise physical measurements we become amazed at the extreme sensitivity of normal ears compared with the extreme insensitiveness of abnormal ears; thus it may require a sound energy as much as 50,000,000 times above the normal pitch for the patient with nerve deficiencies to detect, say, a pitch of 4,000 d. v.

"A large number of curves have been taken on abnormal ears, and these curves have been correlated with the otologic findings. Internal ear deafness (nerve deafness) shows a depression in the higher frequency range (say 3,000 d. v. and above), without changing the sensitivity at the lower frequencies.

"Curves for middle ear deafness involve primarily the lower ranges and are characterized by a larger decrease in hearing for the lower tones, near 1,000 d. v., than for the higher tones, say above 3,000 d. v. The patients may have serious internal ear deafness, with little or no knowldege of it, but middle ear deafness, since it reduces the sensitivity for tones important for speech perception, makes one conscious of trouble as soon as the middle ear begins to be involved. In diseases involving the middle and internal ear it is difficult to say how much of the deafness is due to the middle ear and how much is due to the internal ear.

"Theoretical Bearings.—The theoretical bearing of the data so obtained has been discussed elsewhere. (Wilson and Minton: The Proceedings of the Chicago Institute of Medicine, pp. 157-171, 1921; Minton, John P., Proceedings of the National Academy of Arts and Sciences, September, 1921; Physical Review for 1922.) It is sufficient here to say that by correlating the data so obtained in a study of both normal and abnormal ears much light has been thrown on the manner in which the ear appears to function physically."

DISCUSSION.

Mr. Verne O. Knudsen, department of physics, University of Chicago, opened the discussion and said that a telephone receiver energized by a current from a vacuum tube oscillator is used as a source of sound for determining the

sensibility of the ear to small differences of loudness and pitch. Tones varying in frequency from 30 d. v. to 20,000 d. v. are produced by the oscillator. By means of a divided resistance circuit the intensity of the tones can be varied by any desirable and measurable intervals from the threshold values up to very high values.

Some auxiliary experiments showed that the acoustic energy developed by the receiver diaphragm is directly proportional to the electrical energy which actuates it. The electrical energy is therefore a convenient measure of the relative acoustic energy developed by the receiver diaphragm.

Data that have been taken thus far on a limited number of ears show that the sensibility of the ear to small differences of intensity, measured by the ratio of the smallest perceptible increment in energy to the total energy, $\Delta E/E$, is—

1. Dependent upon the intensity. The ratio $\Delta E/E$ decreases as the intensity increases. For a wide range of moderate and high frequencies the sensibility is nearly constant. Its value for ordinary frequencies is roughly 0.10.
2. Nearly independent of the frequency between 100 d. v. and 4,000 d. v.
3. Nearly the same for all normal ears.

The sensibility of the ear to small differences of frequency, measured by the ratio of the smallest perceptible increment of frequency to the whole frequency, is—

1. Dependent upon the intensity.
2. Dependent upon the frequency. The sensibility ratio decreases from about 0.01 at 50 d. v. to about 0.002 at 1,000 d. v. (for a single ear). For higher frequencies the ratio increases.
3. Dependent upon the mode of reception. Binaural reception yields a resolving power which is about twice as high as the resolving power of a single ear.
4. Only approximately the same for different individuals.

From these data it may be inferred that the average normal ear can distinguish about 400 graduates of loudness (for pure tones of moderate loudness), and over a frequency range of ten octaves (20 d. v. to 20,000) the average pair of ears can distinguish about 5,000 musical intervals or gradations of pitch.

Dr. J. Holinger asked how the flexible tubes influenced the transmissibility of the sound in the hearing device, and whether the smoothness or roughness of the inside of the tube had any influence.

Dr. Sabine, replying to Dr. Holinger, said that the tests as he made them did not bear directly on that point. Theoretically the fact that the tube is flexible would tend to reduce somewhat the intensity of the sound which it transmitted, but that would be a slight reduction, in the order of 50 or 100 per cent, whereas the multiplication would be two or perhaps three hundred fold, so the flexibility of the speaking tube was not an objection to it. Theoretically the smoothness or roughness would have some influence but this effect would be relatively small.

Dr. Alfred Lewy recalled one case which was a stapes ankylosis which presented some very obscure labyrinthine symptoms. The labyrinthine symptoms were not clear cut, and neither were the tests of the hearing clear enough to be certain of the diagnosis. Mr. Minton's diagnosis was spongification of the capsule, and he wondered whether this had not affected the labyrinth sufficiently to interfere with the function of the vestibular nerve. So far as they could tell this was what happened.

Dr. Lewy thought the greatest practical value of Mr. Minton's work rested in the check that could be maintained on the improvement under treatment. The ordinary tests were not sufficiently delicate to determine whether the patients are being helped or not, and he considered this work of great practical value in this way.

In Mr. Knudsen's work, his findings with his much more accurate instrument corresponded almost exactly with Dr. Lewy's experience with the monochord. He had one man, a trained musician, who seemed to hear up to 22,000 He was going to ask Mr. Minton to see this patient and ascertain if he really heard that high.

Dr. Sabine said that in cases of incipient otosclerosis it had occurred to him that a remedial effect might be obtained by stimulation of the ear by intense sounds. For example, in cases where there was some deafness he thought one might stop the progress of the defect by exercise with sounds, and

asked the opinion of the members as to whether there was any rational basis for this belief.

Dr. Sonnenschein said that in the minds of the general practitioners, not the trained otologists, the idea of otosclerosis was that of an actual sclerosis, but this was a misnomer. The condition is really a spongification of the bone and its replacement by a very vascular bone, which interferes with the movement of the stapes. Many methods and devices have been used without avail to overcome this condition, and he wished someone would discuss the effect in those cases where a great deal of vascularization is already present

Dr. Joseph C. Beck was very much interested in the presentation of the subject. The point about the pathologic change in otosclerosis and other conditions around the footplate of the stapes seemed to be not pertinent to the otosclerosis but to other changes of inflammatory character whereby the stimulation by sound would be of value. He thought all would agree that the difficulty with many people possibly is that very early, when they hear but do not understand, they get into the habit of not listening, and so by stimulating them, by insisting upon their listening to tuning forks both by bone and air conduction, this would be of considerable value in the absorption of the inflammatory by-products.

Dr. Beck also called attention to the commercial houses that grasp such scientific devices as this, and that there is an oscillator for therapeutic purposes making a buzzing sound for producing hyperemia and absorption. In the East this appliance is being used largely, and some of the good men in that locality claim that they get results. A good deal had been mentioned about the oscillator at the last meeting of the Academy, and since then he had received a number of letters asking where such a device could be obtained. It seems that people generally are after some device for improving the hearing. In his practice he recommends devices, particularly those of an electrical nature, and thought that people get a great deal of benefit from them. Perhaps this is true only in one small area, as borne out by the charts, but he believed the use of devices for stimulating and improving the hearing, such as the phonograph and other means, were of great benefit. In the otosclerosis, in which we recognize the spongification and

encroachment, this stimulation would not be in place, but in other inflammatory conditions he believed it would be of value.

Dr. A. A. Hayden said he had been intensely interested in the papers and in the subject because of the fact that at least two members of his own family, his mother and an aunt, have otosclerosis. It has been his privilege to view rather intimately the feelings with which these people take up artificial means of hearing. While his mother can hear very well with an electric hearing device, and while many of his patients are greatly aided by these electrical devices as well as by other aids to hearing, speaking tubes, etc., he finds great reluctance on the part of nearly all these people to make use of these devices on account of the rather conspicuous appearance. He did not know how many cases of otosclerosis he sees in a year but thought it was the exceptional case who uses a device and the exceptional individual with nerve deafness who uses a speaking tube. The very first speaking tube he ever was instrumental in having a patient buy was just two weeks ago. It appeared that the people who are hard of hearing would rather keep their defect than use the external devices. This is a strange but true fact.

As to Dr. Holinger's remarks regarding spongification in deafmutes, he was interested to know if that was not a coincidence and not the underlying pathology. It would be perfectly possible for a mute to have a spongification engrafted upon the essential cause for his deafness, just as this evening it might me that the patient shown by Dr. Boot had something other than the cause suggested by Dr. Boot.

In regard to the exercise of the function of hearing with the idea of improving it, Dr. Hayden thought there were a number of facts that militated against it. The presence of boilermaker's disease must not be forgotten, and it must be remembered that that was the result of excessive noise. However, that was not a case in point. He did not believe that the use of the cochlea could increase its efficiency any more than the use of the retina with a hemorrhage would increase the individual's perception of light in that portion of the retina. It is exactly like looking for relief from paralysis in the eye from a muscle that does not functionate. He looked with the

utmost skepticism for relief of loss of hearing from exercising with sound.

Dr. Minton stated that in certain patients who had middle and internal ear deafness he had noticed that stimulation with sounds at certain pitches had brought about sufficient temporary improvement to enable the patient to detect a sound about one-fourth as intense as before. They might require a relative receiver current of perhaps 1,000 initially, but while testing the patient this value may decrease to perhaps 500. This improvement is but temporary, for when the patient returns for testing the next time, the original value—e. g., 1,000—is obtained. It is not likely that we can obtain any permanent improvement in hearing by this means.

Dr. Paul E. Sabine thought Dr. Shambaugh had made the statement that in cases of nerve deafness the speaking tube might prove helpful, whereas in cases of middle ear defect the electrical device would be helpful. The physics of that puzzled him, but he noticed that other men referred to the same thing and wished to know if it was a well authenticated fact.

Dr. Holinger said that he knew of many instances of middle ear deafness where the patient would not part with the electrical apparatus for a million dollars, whereas an elderly gentleman with nerve deafness had tried many of them and thrown them away.

He drew attention to the interesting cases of diplacusis, where the patient perceived a certain sound in different pitch with the two ears. He tested an organ player who had to give up his profession on account of diplacusis, which rendered his playing to himself and the community painfully dissonant. Dr. Holinger tried to produce consonants by varying the pitch of the forks before both ears, and found a variance in pitch of two to six tones in the two ears.

Dr. Sabine thought this might be due to the fact that the maximum sensitiveness of the ear occurs normally for those tones for which the telephone is most effective, and for that reason the patient with nerve deafness would not be benefited by the large amplifications of these tones.

In one patient he had found the defect mentioned by Dr. Holinger and, since the deafness came on, even though she

could hear music, it was very unsatisfactory. There was apparently a difference in pitch of the same tone when heard in the two ears. There might be a possible explanation if a difference in intensity is ever interpreted as a difference in pitch.

MR. VERNE KNUDSEN stated that one of the students at the university had told him that the sustained tones of the violins at the Tivoli were heard differently by the two ears. Examination in the laboratory proved that the same tones were heard a semitone higher by his right ear than by his left ear. His left ear was probably the affected one, since his pitch identification with his right ear agreed with normal ear identification.

Two or three weeks later he noticed that the effect had disappeared, and five months later laboratory tests showed that he heard normally with either ear.

DR. MINTON thought the explanation of the difference possibly was because the two ears may possess quite different sensitivities, and if the tones are not absolutely pure a patient may hear a certain tone with the good ear and quite a different one with the poor ear, since, near the threshold of audition the sense of pitch vanishes and it is impossible to make accurate judgment of pitch difference near this limit. For tones whose intensity is much above the threshold value it is absolutely necessary first to adjust the intensity so that the tones are heard with equal loudness when listening with one ear and then with the other one. When this adjustment is made one can then test to see if a tone sounds of the same pitch in both ears. These requisites make it practically impossible for the otologists to secure any reliable data on the so-called phenomenon of diplacusis.

DR. GEO. W. BOOT thought that the very fact that diplacusis occurs was one of the best evidences that there is a physical resonator in the ear. If the portion of the resonator, for instance, that responds to C^2 normally, gets weighted by some exudate, it no longer responds to C^2 but to some tone lower in pitch. This explains the situation that occurs in diplacusis, for the lower pitch, say C^1, causes the resonator on one side that normally responds to C^1 to functionate, and on the other side the resonator that normally responds to C^2, so that the tone C^1 is heard as C^1 and C^2 at the same time.

With regard to exercising the hearing in otosclerosis to cause the return of normal function, Dr. Boot was reminded of a horse that he once had that was the victim of a bone spavin. The pathology was slightly different, but there was exostosis present in the horse's joint as much as there is around the footplate of the stapes in otosclerosis. After exercising the horse for a short time the lameness would wear off and the horse would seem better, but the next time the horse was exercised he was lamer than before. It was just so with the ear in otosclerosis. If the ear was massaged there was improvement for a half hour and then the condition was worse than before. Not long ago he had a patient in an advanced stage of otosclerosis. She received a blow on the head and at once her hearing became very much better, to the temporary discredit of the doctor who had given an unfavorable prognosis, but in less than two weeks her hearing was as bad as ever.

With regard to hearing instruments, Dr. Boot believed patients with nerve deafness were not apt to be much benefited by the electrical instruments. Patients with fixation of the stapes and increased bone conduction were apt to hear better, for the instrument rests firmly against the side of the head and the sound waves can get in by bone conduction.

Dr. Minton was of the opinion that the mechanical theories of the phenomena of audition are wholly unsatisfactory and are rejected by the physicists, who based their conclusions upon precise physical measurements. Otologists usually believe that deaf people hear with a telephone against the ear because of bone conduction. There is no physical basis for such a belief. Numerous tests have been made to detect "bone conduction" when a receiver is placed on the ear. Negative results are always obtained. The real explanation is that the telephone, when placed over the ear, radiates all its sound energy directly into the auditory meatus and little or none of the sound energy is lost to the surrounding space.

Dr. J. Gordon Wilson said that during the war he observed a great many men made deaf by shell concussion (explosion). A number of these men were improved by stimulating the auditory tract by pure tones conducted through the mastoid bone if very deaf, or through the air if not so deaf. They im-

proved to a considerable degree, but not entirely, and kept this improvement. This subject was discussed in a lecture before the Harvey Society in New York in 1917 and published in their series of lectures.

DR. SONNENSCHEIN emphasized the fact that it is very important for otologists to know more about acoustics and the diagnosis of such symptoms coming within their sphere. He thanked the gentlemen for coming and enlightening the society. He thought one of the most practical points made was the fact that the old time-honored "garden variety" of amplifying the sound by placing the hand behind the ear seemed to give as much relief as the modern metallic devices.

Another interesting point was with reference to the small amount of energy required to produce audition, as shown by a 40 watt light having enough energy to activate a string of receivers going about 25,000 times around the earth.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday Evening, January 9, 1922.

THE PRESIDENT, DR. ROBERT SONNENSCHEIN, IN THE CHAIR.

Pulsating Sphenoid.

DR. CHARLES H. LONG presented a case of pulsating sphenoid and said his reasons for presenting this patient were twofold. First, he discovered that he had escaped a bad result from the treatment; second, he discovered that the man had a pulsating sphenoid. The patient was a man of 59 years, who was referred to him because of deafness and tinnitus in the right ear. Examination revealed an ethmoiditis with a mucopurulent discharge down the middle turbinate. About the 19th of December he exenterated the ethmoid, and along the anterior third of the turbinate was a mass of granulations toward the frontal sinus. He opened the frontal cells, and when the middle turbinate was removed with the ethmoid cells he noticed a discharge from the sphenoid. He wiped this out and there was a severe reaction. The man had a badly developed septum from a former traumatism. The following day the right eye was projected and swollen, red and congested. He spent several sleepless nights following the operation and had considerable pain, not only in the back of the head but through the eye. The ordinary treatment was used—washing out the nose with saline and using a hot water bag to the eye. About a week after the operation he syringed the sphenoid and the reaction from this put the patient back to bed. He reported at the office two days later, and it was then that he had a pulsating sphenoid.

The cause of the pulsation was not altogether clear, and it was not clear why he got projection of the eye and cellulitis, unless the membrane between the orbit and nose was perforated, or else in the sphenoid there was no bony membrane between that and the internal carotid, or else there was necrosis of the external portion of the sphenoid and possibly some pulsation of the brain or a tumor. There was no pain and the hearing in the left ear was normal. In the right ear he could

hear an ordinary conversational voice at ten feet and the whispered voice at four feet.

DR. G. W. BOOT asked if the carotid was bare in the cavity.

DR. LONG replied that he did not know. After having wiped the cavity out he was satisfied to leave it alone.

DR. EDWARD P. NORCROSS presented a paper entitled:

"A Case of Acute Mastoiditis with Unusual Complications."*

*See page 501.

DISCUSSION.

DR. JOSEPH C. BECK recalled a case of an abscess occurring in the side opposite to the mastoid operation. The patient gave absolutely no symptoms referable to a temporosphenoidal abscess in the side opposite the operated side, but died suddenly a week after the mastoid operation. Postmortem examination showed that it was an old abscess. The lining of the abscess cavity was very thick, and he thought probably the operation activated this old abscess and caused a direct extension into the region of the fourth ventricle.

DR. G. W. BOOT thought a good deal of light might be obtained by means of a lumbar puncture, and asked what the lumbar puncture in this case showed.

DR. OTTO STEIN considered it a rather unusual complication and thought there were very few cases reported. When Dr. Dean reported his operation for a retropharyngeal abscess by the external method before the society, in discussing the operation Dr. Stein reported a case under his care similar to the one just reported by Dr. Norcross. He believed such cases occasionally occurred and were probably not recognized, but thought Dr. Norcross' case was a rather rare type of retropharyngeal abscess.

DR. ROBERT SONNENSCHEIN reported a case seen a month and a half ago, in an eight months old infant that developed a right sided otitis media. The right drum membrane was incised and profuse discharge escaped. Shortly afterward a retrotonsillar abscess developed on the right side. After this was incised a similar abscess appeared on the left, which also was opened, and following this another reappeared on the right side. Then the right cervical lymph glands, which had been swollen during the whole illness, broke down, were in-

cised and the patient recovered. Streptococcus hemolyticus was found in the pus contained in the glands.

Dr. Norcross (closing the discussion) said that the spinal fluid was under slightly increased pressure. The cell count was 10 and the various tests were all negative. Roentgen examination of the mastoid, spine and skull showed nothing abnormal.

Tooth in the Right Bronchus.

Dr. Harry L. Pollock presented a young lady with a history of having a tooth in the right bronchus.

On June 30, 1921, this patient had a tonsillectomy performed in a small city in Ohio. The operation was performed under general anesthesia, with the patient's head in the operator's lap. The open method of anesthesia was employed first, then the blowing method, suction being employed. Just before the operation the patient warned the doctor that she had a left cuspid tooth on a pivot, and immediately after the operation she discovered that the tooth was missing. She called attention to this fact, and the physician told her that the operating room had been cleaned and the tooth had probably been swept up and thrown away. She recovered from the tonsillectomy within a few days and went to her home in Minnesota, and there developed a right sided pneumonia. She recovered from this, but still had symptoms of cough and pain in the chest. She was then taken to another hospital and told that she had tuberculosis and was treated for this condition. She continued to have symptoms of tuberculosis, but they were unable to find any bacilli. She was then sent to St. Paul, and for the first time a radiogram was made, which revealed the tooth in the right bronchus.

She then came to Chicago and was seen by Drs. Pollock and Beck, this being about five months after the tooth became imbedded in the bronchus, and Dr. Edwin McGinnis was called in to remove the tooth. The patient was put under Gwathmey synergistic anesthesia, consisting of three hypodermic injections of $\frac{1}{8}$ grain morphin in two cc. of a 25 per cent magnesium sulphate solution, one-half hour apart. Following the last hypodermic three ounces of ether, with three drams of paraldehyde in three ounces of olive oil, was introduced

into the rectum. The magnesium has a synergistic effect on the morphin and increases its effect greatly without affecting the danger of the morphin. In these cases it is almost impossible to give a general anesthesia on account of the infection and the pus. The radiogram showed a distinct abscess in the lung. Dr. McGinnis passed a bronchoscope and found the abscess cavity, but could not reach the tooth. It was then decided to let some of the inflammation subside and then attempt it again, but on the following day the patient coughed the tooth out. She developed a little pleurisy on the right side and a little more infiltration but had now completely recovered. The operation was performed three weeks previously and they were sure that had the capsule not been opened the tooth would not have been expelled.

This case was presented to emphasize the dangers of tonsillectomy and the fact that this patient had gone for five and a half months with a history of having lost a tooth, with pneumonia and socalled tuberculosis, before the radiogram was taken which disclosed the tooth in the bronchus.

Primary Lateral Sinus Thrombosis of Jugular Without Apparent Otitis Media.

Dr. H. Pollock presented a boy who was first seen on March 10, 1921. He had then had a cold and pain in the left ear for about a week. A moderate degree of otitis media was present. The child was very unruly. The mother was advised to keep him in the hospital, but he would not stay. He was reported to be better the following day, but two days later the pain was worse and he was again brought to the hospital. Upon the second examination the ear was still red, there was no bulging of the membrane, and hearing was poor. The temperature was 100 to 101 degrees F. He was kept in the hospital and hot compresses were applied. The second day afterward a thick, hard, painful swelling was noticed down the neck, and a diagnosis of lateral sinus thrombosis, with extension to the jugular, was made. The following day the sinus was opened, the thrombus removed and the jugular was ligated. The boy made an uneventful recovery. There was never a perforation of the ear drum nor any discharge from the ear. The ear was red but there was never any bulg-

ing. There was very little pus in the antrum, but it went through the middle ear into the lateral sinus. The boy's hearing at time of presentation was perfect.

In this case the Beck-Crowe test, made by compressing the opposite jugular, gave a positive result. There was great dilatation of the blood vessels on both sides. There were no symptoms of meningitis. The jugular was not dissected, but simply ligated down low. A small abscess formed, which was drained two days later. The wound was left wide open and permitted to granulate in.

The morning of the operation the temperature ran up to 102 to 104 degrees F. As soon as the operation was finished, although the bed was prepared with hot bottles and blankets, the boy had a terrific chill just as he came out of the anesthesia. The temperature never went up following this, but remained practically normal the rest of the time.

Atrophic Rhinitis.

DR. H. POLLOCK said that there was nothing unusual in the diagnosis of this case. The disease had lasted for several years. Dr. Beck had performed a bilateral ethmoid exenteration. Following recovery from this she decided to go further with the treatment and have the place left by the exenteration fill in. They always do the ethmoidectomy by the Mosher method, with the middle turbinate intact. There was pronounced atrophy on both sides. They then implanted into the septum another septum from a previously operated case.

In this work they had carried out a series of experiments previously in trying to fill in this space, starting with paraffin. This proved unsatisfactory in most cases, and they then tried other substances. The first thing tried out was the implantation of fascia lata. This was satisfactory except that it was so small. In the first case they took both fascia latas and implanted them on one side, but they shrunk to almost nothing. Following this they tried fascia from another individual, but had very little success with this method. Most of it sloughed out. They then tried another method of doing a submucous operation on one side and then implanting the substance between the bony and cartilaginous septum. It does no good to put the implant up so high, because the atrophic condition is

down low. They next tried the plug that has been used in osteomalacia, consisting of paraffin, iodoform and wax. This makes a hard substance that has been used in osteomyelitis, but in their work it did not prove satisfactory. It all sloughed out. So they resorted to the implantation of another septum from a previously operated case. They have done this in probably thirty cases and only one or two have sloughed out. The rest have healed beautifully. At first they tried to do both sides at once, but found that it took away the nutrition from the septum to remove both flaps, a small perforation followed and bone and cartilage sloughed out, so they now do one side at a time.

This patient was operated on the right side about three months previously and immediately showed improvement. The odor disappeared entirely, but the other side became worse. They then operated on the left side, and at present there was bulging on both sides.

It is sometimes necessary to use two septums in this operation. On the right side they made an incision far to the back so they could make one in front and dissect up the perichondrium and implant a second septum if it proved necessary.

The result was lessening of the discharge and disappearance of the odor. It had been a year and a half since they operated the first case and with one or two exceptions they had no infection.

DISCUSSION.

DR. H. R. BOETTCHER asked if they closed the initial incision.

DR. POLLOCK replied that they used a little collodion and cotton and a Burnay split was then kept in for twenty-four hours. The fascia was not used for a few hours after removal, but in all these cases operated after the primary ethmoidectomy they waited until they got a submucous that they knew to be free from infection and then implanted it within five minutes.

DR. JOHN A. CAVANAUGH stated that he had operated upon several cases, using the method advocated by Dr. Pollock, but his results were not as satisfactory.

Dr. Cavanaugh transplanted a large piece of cartilage and bone removed from another case. It grew in very well and the patient progressed for a time, but the secretions soon returned. About a year later the man developed a severe nasal hemorrhage and consulted another physician during Dr. Cavanaugh's absence from the city. This physician told the patient the operation was poorly done and in order to get relief he would have to undergo another septum operation.

Pulsating Artery in the Left Pharyngeal Wall.

Dr. H. C. Pollock presented a woman with tinnitus in the right ear and right sided otitis media. Examination of the nose and throat showed marked pulsation behind the posterior pillar, with a pronounced bruit on the left side. He believed this was not an anomalous artery but an aneurysm on the left side with an otitis media on the left. The tinnitus was probably due to the otitis media.

Pituitary Dystrophy.

Dr. Joseph C. Beck presented a child, boy, aged nine years, who was almost completely blind. There was a history of severe headaches, vomiting and all symptoms of brain tumor. The child was very fat and entirely unable to walk. He presented the typical Froelich syndrome of pituitary dystrophy. The blindness had developed rapidly, with a sudden disappearance of the pressure symptoms, nausea, vomiting and headache. The intellect was not impaired at all. The child answered questions freely and sang and recited in an almost precocious way. He could not walk, as stated above, because his feet seemed unable to bear his weight.

By means of X-ray and other findings, they made a diagnosis of pituitary tumor, which had broken down into the sphenoid sinus region without obstructing the breathing, and they decided to perform a subtemporal decompression to see what result would be obtained. Light perception was restored, but he was still unable to walk.

Dr. Beck asked for an expression of opinion as to whether they should now do an intranasal operation, or should they do nothing more.

In reply to a question as to why he did not use radium, Dr. Beck said he had not thought of trying it in this case. He

believed ductless gland therapy would be of no avail and thought the only question was whether further operation should be attempted. A neurologist had expressed the opinion that there was nothing to be done and concurred in the diagnosis.

(Note—Since the above presentation before the Chicago Otological and Laryngological Society, Dr. Beck operated upon the boy by way of the cranial route and found no evidence of a tumor in the region of the hypophysis. The boy appeared to have very much distended lateral ventricles. He did very poorly after the operation and two weeks later died from symptoms of a low grade meningitis. Postmortem showed that this was true. It further showed that he had no changes in the sella turcica. He had, however, a marked internal hydrocephalus.)

Carcinoma of the Larynx; Laryngectomy.

Dr. Joseph C. Beck next presented a man with a history of a growth in the larynx which had persisted for several months. This was treated by a local specialist, but he was finally referred to a large private clinic, where a laryngectomy was performed and was subsequently rayed with large doses of radium emanations. There was a recurrence and he was given more large doses of radium emanations and was then told that was all that could be done. The patient was then referred to Drs. Beck and Pollock because of choking sensations, and Dr. Pollock did a hurried tracheotomy. Following this they fed him up for one week or ten days, until he was in condition for a laryngectomy.

One of the interesting features about the case was that the operation was done under what is termed the synergistic anesthesia of Gwathmey. Dr. Beck reported fifty cases operated upon under this synergistic anesthesia, and exhibited a chart of the cases. The operations were the ordinary ones of the nose and throat, as, for instance, tonsils, septum, turbinectomy and ethmoidal exenterations, as well as the borderline and major operations about the head and neck. He stated without hesitation that he had never tried anything in the way of an anesthetic that worked so beautifully as this does. He had performed plastic operations without any pain whatever. They

had sometimes found it necessary to give some additional ether or gas, as Gwathmey had stated in his articles. He believed there was no danger of toxic effects from this method and that there were very few untoward effects of any kind. In one case there was some irritation of the rectum for a day or two, and in one case mild infectious abscesses at side of injection developed. They had not used this method in children, but for a general anesthesia, like a sinus operation, where the patient could be kept sufficiently conscious to be able to answer questions, blow the nose, etc., he had never seen anything to equal it. In the near future they were going to publish their report on the first one hundred cases, giving the original technic as well as their modifications. At this time they referred anyone interested to Gwathmey's articles, which appeared in the A. M. A. Journal, or to Dr. Bernheimer, who has had this work in charge.

In the patient presented the operation was done according to the Glück method. They found that the growth was entirely intralaryngeal, not extending to the outside at all, and he felt that there would be no recurrence. They had used no radium and would not as long as things remained in normal condition. The man had developed erysipelas in the course of the treatment, from which he completely recovered.

Another interesting thing about the case was the matter of feeding, which was done by an Ewald duodenal tube in the beginning, but the patient was now able to take ice cream and some other things without the tube.

Hyperesthetic Rhinitis.

Dr. J. C. Beck presented this patient because of the work which had been done by Dr. Arnold B. Kaufman in this disorder. The treatment of hyperesthetic rhinitis was considered practically hopeless accordng to old methods. He asked that the privilege of the floor be extended to Dr. Kaufman, so that he might speak of his treatment of these interesting and difficult cases.

Paraffinoma.

Dr. Harry L. Pollock presented a patient who had sustained a broken nose, which resulted in a "saddle nose." A surgeon had endeavored to remedy this condition by injecting paraffin and the patient now had a real paraffinoma. The nose

was red and in worse condition than before this operation. The skin was infiltrated and the growth was adherent to the tissues. They intended to remove the entire mass, right down to the nasal bone, and do a plastic operation, as these tumors frequently become malignant.

DISCUSSION.

Dr. A. B. Kauffman said the case presented was one of a series of types in which during the past year they had attempted to determine what part, if any, deficiency disease may play in producing any pathologic state or malfunctioning of parts in the ear, nose and throat other than fertile soil for the growth of saprophytes or pathogenic organisms. In animals it has long been recognized that the snuffles of hogs is primarily a nutritional disorder, and Hess has long called attention to the frequent coincidence of nasal diphtheria and latent or subacute scurvy. They made an effort to observe the possible relationship of dietary deficiencies, particularly vitamin deficiencies, to various nasal and aural conditions which were of more or less unknown etiology and which pathologically showed changes not of an inflammatory nature nor directly due to bacterial invasion. Of these, hyperplastic ethmoiditis and otosclerosis appeared to be the most conspicuous.

In hyperplastic ethmoiditis, histologically, they found the osseous changes were those of absorption, the bone cystic or thickened by a rarefying process. These pathologic changes are not unlike those found in other well defined deficiency diseases, so on this hypothetical basis they have given to one group of typical cases a dietary rich in water soluble B and C vitamins, while in the second group there was simply added to their dietary cod liver oil, which is abundant in the fat soluble A vitamin. The first group showed no particular improvement, while the second group began to show definite amelioration of symptoms. Therefore, on the basis that in this condition there may be a deficiency in the fat soluble vitamin, they had determined the inorganic phosphorus content of the blood because of the fact that investigators at Johns Hopkins Hospital gave sufficient basis to assume that the level of blood phosphates is determined in part by the amount of fat soluble A vitamin available for the body needs, and their

results have shown for the most part a diminution in the blood phosphate content.

In otosclerosis they had more basis for the assumption that the condition may be a manifestation of a deficiency disease because of the analogy of the pathology in rickets, and in this condition it must be borne in mind that in rickets, although a condition of early infancy and childhood affecting young bones, there are definite changes in the temporal bone. Mayer in his recent book on otosclerosis studied the changes in the temporal bone in a nine months old infant with well advanced rickets. He found a definite bony degeneration in the cartilage about the oval window, and that the newly formed bony tissue in the region of the oval window remained uncalcified, the defect being a lack of calcification rather than the formation of cells.

Here, then, in rickets, a disease of infancy and childhood, and otosclerosis, a condition existing between the twentieth and fiftieth year of life, changes occur in the temporal bone that are not dissimilar, although their end-results are not quite identical. Furthermore, it is known that rickets is essentially a dietetic disorder, centered on the röle of vitamins and more specifically on the fat soluble A vitamin; that it can be produced experimentally by diets deficient in this vitamin, and has been cured and prevented by the administration of cod liver oil, which contains the deficient accessory.

Can it not then be assumed as a working hypothesis that a similar deficiency, acting throughout a different period of life, may not produce such a clearcut disorder as in infantile rickets, but bony changes elsewhere? There are likewise certain blood findings in active rickets, the most important being a lowered content of inorganic phosphorus. This finding is considered as nearly conclusive evidence of active rickets. In some of these cases of progressive otosclerosis they have likewise found a diminution of the blood phosphate, and as mentioned before, this level is determined in part by the amount of fat soluble A vitamin available for the needs of the organism. They also hope to show changes in the temporal bone of animals which have been fed on diets deficient in the food accessories similar to those found in otosclerosis.

Dr. Charles M. Robertson said that he had contended that rickets and otosclerosis were practically the same thing.

In Dr. Beck's case of pituitary pressure, Dr. Robertson thought he would be decidedly justified in operating upon the case, with the probability of finding a cyst rather than a tumor. The history of the case would seem to indicate that a cyst was forming, because of the rapidity of the increase in size. If a solid growth had formed with such rapidity the child would have been dead. If the growth was a cyst the prognosis was favorable as regards sight and life. Dr. Robertson had reported two cases of cysts of the pituitary, operated intranasally. In one case the vision was reduced to almost zero, and the optic nerve looked chalky, like a profound optic atrophy. When pressure was removed the vision came back to normal and the optic nerve assumed normal appearance. He thought to let the case go as at present meant nothing but disaster, and the child would be better off dead than in the present condition. He was in favor of operation on the chance that the growth was a cyst underneath the pituitary, and thought it would be crowded down so that it could be extirpated through the sphenoid sinus.

In the first case of carcinoma of the larynx, he thought the gentleman was very fortunate. He had a unilateral growth, which was taken out by the only method—from below upward. This is the easiest method of removing the larynx and the safest. One point in removal of the larynx is vital: The recurrent laryngeal nerve running up the other side toward the larynx is very easily irritated. Even if rubbed slightly it would irritate, and as the fibers of the pneumogastric are very intimately associated with the recurrent laryngeal nerve, respiratory paralysis may occur from irritation of these fibers. The fact that the patient had an erysipelas was in his favor. Dr. Robertson had seen two cases, one in the Royal Hospital of London and one case in private practice, where erysipelas cured such a growth. This fact was so well known that patients have been infected with erysipelas in order to promote cure. In one case seen in London the growth affected the larynx and the pharynx. When he examined the patient after having had erysipelas the contour of the larynx and the pharynx was as nearly normal as he ever expected to see them.

It was impossible to tell that the patient had had a tumor of malignant or any other type.

In his opinion about 40 per cent of the success in cases of laryngectomy depends upon the operator, and the other 60 per cent upon proper after-treatment. Patients die, not because they are not operated upon properly, but because they are in hospitals that are not taking care of such cases as a routine thing, thus prohibiting the proper care and after-treatment. This work needs a staff specially trained to take care of this type of case. They have to be kept clean, cleaned every half hour if necessary. The discharge that comes down from the throat is excessive, and if it gets into the tracheal wound there will be sloughing. He considered the operation of laryngeal fissure much easier. In the case presented, Dr. Robertson thought the man would recover a voice that would be husky, but one that would permit him to talk for hours, if necessary. He had always made it a practice to broil these cases with a hot iron after the diseased tissue was removed. Then the cicatrix forms a straight band across the larynx as a fibrous band.

Regarding the use of radium, Dr. Robertson expressed the opinion that it had killed ten times more people than it had even benefited, but what the future will develop cannot be told at present. He had seen many cases which radium had helped for a few weeks, and then the growth spread rapidly. In his service at the Alexian Brothers Hospital, where there were many cancers of the throat, he had yet to see any except the superficial ones that had been cured by radium or even alleviated. Outside of the extirpation of the tumor in toto, he believed there was only one thing to do: In his opinion, the application of actual heat was the thing that destroyed malignancy. It made no difference whether the growth was in the throat or some other part of the body, and if one used the knife he should sear the wound afterward. Doyen, in Paris, uses voltaization and fulguration, which char the tissues and they heal. In cauterization under ether great care must be used or an explosion of the ether gas may result. The first time he ever tried this he was afraid the patient would explode. After opening the trachea, he used the Paquelin cautery, and in order to avoid exploding the patient

he waited until after he had expelled all the air in the lungs and then cauterized before the act of inspiration. He had since seen the cautery used without bad result, but warned of this danger.

Regarding Dr. Pollock's case of sinus thrombosis without rupture of the drum membrane, Dr. Robertson had seen two cases of sinus thrombosis in which there never was discharge in the ear, but at the time of operation the attic cells were full of pus. He did not know whether one was a tubercular case or not, but the drum was either resistant or there was not enough pus to break through by pressure. The suppuration had been going on until the patient was beginning to have profound meningitic symptoms. The eardrum membrane was not even pink, and yet it was an old case of cholesteatoma.

Dr. G. W. Boot said he had had several cases that showed peculiar lung symptoms. In one case the child had inhaled a toy arrowhead. Another patient had inhaled a collar button, which had remained in the lung for two years before a diagnosis was made and at that time an abscess had formed. A third case, a boy, of about fourteen years, had irregular symptoms of tuberculosis, and a radiogram showed a piece of wire in the lung. The symptoms dated from about the time of a tonsillectomy and the wire was probably from a tonsil snare.

Dr. Boot thought it strange that general practitioners were so slow about taking X-ray pictures in peculiar pneumonia cases.

In his opinion the three great contributory causes of carcinoma of the mouth and throat are a dirty mouth, syphilis and tobacco.

Dr. Harry Kahn thought that Dr. Kaufman's recent work was epochmaking. He knew the results obtained in some of the cases treated were remarkable. There had recently been discussion in the Journal of the A. M. A. as to rickets as a vitamin deficiency disease. Some English believe there is an antirachitic vitamin. The Americans do not take this so seriously, and Hess has stated that the A vitamin is not a necessary article of food in the treatment of rickets and recommended the application of sunlight in order to prevent rickets. In the same paper a table of percentage of blood phosphates, both in normal and rachitic children, is given. H. Steenbock

(Science 50, 352-1919) made an interesting generalization in regard to fat soluble A vitamin. All food containing fat soluble A vitamin is usually yellow in color—butter, carrots, sweet potatoes, yellow tomatoes, corn, etc., and should form a part of the diet.

DR. ROBERT SONNENSCHEIN asked whether it was not a fact that unboiled milk contained almost as much fat soluble A vitamin as most any other substance.

Secondly, whether it is not the organic iodin found in cod liver oil that is partly responsible for the good effects. If it were not for the iodin, would not unboiled milk alone be just as good as any other substance?

DR. ARNOLD B. KAUFFMAN, replying to the discussion, said that cod liver oil in otosclerosis is not new. As far back in literature as one could find reports, almost every otologist abroad had used cod liver oil in otosclerosis because of the good results that were obtained, and because of the similarity between otosclerosis and rickets. It was used empirically and no mention was made of the fat soluble A vitamin. As to the English and American viewpoint of cod liver oil in rickets, Hess had come out with sunlight and ultraviolet rays, and most authors admit that they obtain the same results as from cod liver oil, but get the effect from different angles.

In answer to Dr. Sonnenschein's questions, Dr. Kauffman said the cod liver oil contained the fat soluble A vitamin in abundance, particularly the unrefined oil. It is impossible to cure rickety infants with unboiled milk, but cod liver oil will cure them.

Dr. Kauffman cited the fact that when Col. Garrison spoke on gastrointestinal conditions in relation to deficiency disease recently, he stated that during his experience in the Himalayan mountains he had failed to observe anything that he could point out as due to deficiency. He had never seen a case of deafness during his service there. How much importance could be attached to this statement Dr. Kauffman did not know, but it seemed that in various climates where people live on a strictly vegetable diet and uncanned foodstuffs these conditions are apparently absent.

DR. JOSEPH C. BECK, closing the discussion, thanked Dr. Robertson for bringing out some of the points he had not

taken time to emphasize, but which he considered important, but he took exception to his very emphatic statements regarding radium. He agreed that in many cases the results were not good, but felt that such emphatic statements from a man of Dr. Robertson's standing would put a damper on the use of radium before enough time had elapsed. In Dr. Beck's opinion, more people should use radium so that the results can be definitely determined. He had hoped to present a patient for whom they had performed a laryngectomy from within outward by the use of radium. The man had refused operation, but did not refuse the radium therapy. He considered radium a very important adjunct in the treatment of cancer, but at this time could report no cures.

"Conditions for Progress of Otolaryngology."

DR. J. HOLINGER addressed the society on "Conditions for Progress in Oto-Laryngology."

During the last thirty years our knowledge of anatomy, physiology and pathology of the ear has been revolutionized in all its particulars. To these advances America has contributed very little. Dr. Shambaugh has done very creditable work in anatomy of the labyrinth, but besides that little can be shown. It is questionable whether Germany, Austria and Switzerland will be able to continue with this tedious and expensive work as they have done so far. It is time for America to take an active part. The writer has discussed the conditions for doing advanced work with his former chief, Prof. Siebenmann of Basel, Switzerland, and can formulate those conditions under three main heads.

1. A large ear, nose and throat clinic with 10,000 patients a year.
2. Extensive laboratory facilities.
3. A great deal of fresh postmortem material.

All this with a sufficient staff of well paid assistants and a competent leader who can work together for years in connection with graduate and postgraduate teaching.

DR. GEORGE E. SHAMBAUGH was very much pleased to hear Dr. Holinger's remarks, because they brought up subjects which deserve serious thought. Dr. Holinger pictured a situation in regard to the specialty of otolaryngology in Basel.

We have nothing in this country that parallels this situation, and it is not at all likely that our development will be exactly along the same line. We have our own problems which must be solved in our own way. Dr. Shambaugh pointed out that there were some deplorable practices in connection with the treatment of patients suffering from nose and throat conditions at present, which have come about largely from the practice of instructing interns in general medicine and general surgery in the technic of operations in the nose and throat. These men can have no proper appreciation of the indications for these operations, and the result is that we are seeing a great many unnecessary operations by this type of practitioner. In certain ways there has been as much substantial progress in our specialty in this country as there has been abroad, but what has been accomplished has been worked out largely through individual initiative. We have had no assistance in the way of departmental equipment or organization, which is essential in carrying out any sustained piece of research.

There was one phase of the subject which Dr. Holinger did not touch upon, but which is quite vital when considering progress in our specialty, and this is the method employed in training men for this work. In Europe the prospective specialist has always gone through a period in which he served as an apprentice or assistant in a well organized and equipped clinic. Our own men who have sought work abroad have very rarely been able to avail themselves of this plan, which is reserved for their own men. A system of intensive courses, which might well be termed courses in "forced feeding," have been developed to accommodate especially the Americans. Courses of this sort are of value chiefly for those who have had their period of training by serving as assistants in a well run clinic, but they have no proper place for those who are undertaking to learn the subject without that training. The result has been the flooding of this country with a great many men whose training has been a spurious preparation based upon facts ladled out to them in these courses. The real training is acquired only by men taking part in the actual work—that is, serving as clinical assistants. One learns very little by sitting around listening to others tell how they do things.

Dr. Shambaugh thought that the facilities for securing

proper training in otolaryngology were certain to be greatly improved in the near future. The fundamental training must be gotten here in our own institutions. After this has been acquired, courses such as are offered in our postgraduate institutions, as well as in certain foreign cities, might well be taken, but such courses should not be offered as a substitute for the real fundamental training. Those of us who occupy positions in our teaching institutions should bear in mind the difference between the opportunities in our own universities and those that exist, for example, in Basel, and should strive to educate our universities to a proper appreciation of what a department in otolaryngology should be.

Dr. Elmer L. Kenyon said in regard to research being placed on a scientific basis, Dr. Shambaugh had discussed one phase. There were still other phases which might be discussed. In his opinion, Dr. Holinger had struck a keynote which was right, and he thought the speaker had struck it at the right time, for we are on the point of paying salaries to scientific men, even scientific medical men, and this will do much toward placing research on a truly scientific basis.

Dr. Kenyon desired to express two thoughts in the encouragement of Dr. Holinger's suggestion: first, he doubted whether in the history of this country anybody had gone to the State Legislature and said this thing should be done; but we know that where other men in other walks of life have gone to State Legislatures and said that certain things should be done they often have been done. In his opinion, what was needed was a body of men who could establish an organization to set the thing going. Secondly, we occasionally do build great things at once. The Rockefeller Foundation was all built over night, as it were. But as a rule we do not build in that way, but rather with small beginnings, followed by gradual accretions. He believed the thought should be to start things now in the right direction, but not necessarily to expect this generation to put the project on a final basis, but perhaps the next, or even the third generation. We should attempt to set something going that is solid, and look for future generations to build on it. In this way, he believed, the substantial research foundations were more likely to be built up, rather than through some great act fulfilled all at once.

Dr. G. W. Boot agreed with all the speakers and believed there was probably no one particular in which the members were so deficient as in the pathology and histology of the subject. Probably with the exception of Dr. Shambaugh, there were not half a dozen members of this society who had ever cut a section of a temporal bone. We talk very learnedly of otosclerosis being connected with rickets, as the result of focal infection, and the result of syphilis, but we know nothing definite about it. It is all supposition, and we will never know anything definite until something was started such as Dr. Holinger mentioned. If this is not started it will be the same old story—"the fault, dear Brutus, is not in our stars, but in ourselves that we are underlings."

Dr. Boot moved that a committee of six men, members of this society, be appointed to see what could be done in this connection, the committee to consist of the president, the secretary, Dr. Holinger and three others, the object being to get such a foundation started.

Dr. Joseph C. Beck seconded Dr. Boot's motion, and said all were familiar with the political side of the question. Politics played a great röle in the securing of postmortems. All had had experiences with the undertaker politicians who prevented postmortems. They often have to fight for them, even though the families of the patients are willing to have them performed. Dr. Beck has reported this many times. The sentiment is decidedly against postmortems in this country, sometimes because of religious sentiment—this particularly among the Jews, but in the old country it is almost a law that postmortems shall be performed.

Another point is the matter of pathologic investigation. He offered the necessary facilities for this training, but had found very few men who took the necessary interest. They did not see the importance of this side of the subject but were interested only in the operative side, and the men who came for postgraduate work cared nothing for pathology.

Dr. Beck thought an earnest effort should be made by the society to start the thing suggested, and even if it took a hundred years to accomplish it, they should get started.

Dr. Kenyon thought procedures should not be complicated any more than necessary.

Dr. Boot thereupon, with the permission of the seconder, withdrew his motion.

Dr. H. R. Boettcher agreed with Dr. Beck that it is almost impossible to obtain postmortem examinations.

Dr. Harry Kahn thought two factors entered into the question: First, better education of the medical students; second, the apathy and antagonism of the public toward the medical profession. Anything the medical profession brings forth will be combated by all the sects and "isms," and unless they have a large lobby they will not get anywhere. If they have nothing that interests the State Legislature they will get nothing from it. The public has to be educated before such things can be put through. In Europe everywhere the doctor is looked up to and respected and honored.

Dr. Holinger, closing the discussion, thanked the gentlemen for their remarks. He thought the conditions here and abroad were not essentially different. He had seen the whole clinic in Basel grow out of nothing. When he worked with Siebenmann in 1891 the clinic was held in the private consultation rooms.

In regard to the postmortems, he thought they had the same prejudices to fight over there as we have here.

He had not brought this matter up before but had had it in mind for many years. This much is certain, nothing can be accomplished unless we make a start. He believed if the public was educated to the questions that need to be solved it will become interested and will help with funds. Politics and other difficulties can be overcome if the profession will work together. He had brought the matter before the Council of the Chicago Medical Society and felt that when there was unanimity much would be accomplished.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

DR. HILLIARD WOOD, PRESIDENT.

February 20, 1922.

Fracture of the Skull.

DR. HERSCHEL EZELL presented Mr. E. R. K., aet 45, motorman, who gave the following history: Twelve years ago was hit in right temple with hammer, resulting in scar about 1½ inches long, located above and in front of right ear. January 4, 1922, was in street car collision and fell, striking head on register in front part of car. Was not rendered unconscious by blow, but felt dizzy and unsteady following accident. Thirty minutes later had expectoration of blood, with discharge of blood from right ear. Few days later aural discharge became purulent in character and continued so for about one week. Patient complained of deafness, pain, discharge and occasional tinnitus right ear since accident. Alleges that previous to injury the right ear was normal. No trouble alleged in left ear.

Examination showed right ear drum retracted, scarred and discolored: no perforation; no discharge. Watch and tuning forks not heard by either air or bone conduction. The stethoscope test was used in an effort to detect malingering, but no hearing could be demonstrated in right ear. Left ear normal; hearing perfect by both watch and tuning forks. Nose and throat negative. X-ray by Dr. H. S. Shoulders shows fracture 1½ inch long in temporal bone, slightly above and in front of right external auditory canal. It is believed the injury extends to the base, but this could not be demonstrated by X-ray plate.

In presenting this case, Dr. Ezell asked an opinion as to whether this fracture of the skull, as revealed by the X-ray plate, could be the result of the injury some twelve years ago or is the result of the more recent injury.

DISCUSSION.

Dr. W. G. Kennon suggested that Dr. Shoulders, who made the plate, could give more definite information on this point.

Dr. Hilliard Wood, referring to the stethoscope test, said that he had become familiar with this test in examining Army applicants during the war. It was his opinion that this test is of doubtful value unless the rubber tubes are some three or four feet long—i. e., long enough to enable the examiner to stand some distance behind the patient; otherwise the patient will hear with the good ear by air conduction even though the other ear is deaf. He regards the stethoscope test as especially valuable in detecting malingering. He said that another good test, similar to the laerm apparatus test, consisted in irrigating the good ear and talking to the patient during the irrigation, the patient being unable to hear with the good ear during the process of irrigation. Dr. Wood also pointed out that in this type of cases, sooner or later, the question of damages arises, and in the matter of final settlement the doctor is virtually an umpire, as upon his statement the final awards are usually made; so that one cannot be too careful in these cases, in order that full justice may be done, both to the injured person and to the company which pays. He suggested that a Barany test might give valuable information in this case.

Dr. M. M. Cullom regards the noise apparatus test as better than the stethoscope test in detecting malingering. Another valuable test mentioned by him is the closing of the good ear and requiring the patient to count; if deaf in the ear being tested, the voice will be raised.

Dr. Eugene Orr, speaking of the various tests, said that he considered no one test to be absolutely infallible, as his experience both in Army service during the war and with the War Risk Bureau since had been that the majority of the men are coached in advance and are perfectly able to pass any test which may be given. Regarding the fracture, he advised the use of the fluoroscope, as he believes it to be almost impossible to diagnose fracture of the skull accurately except by the fluoroscope. He thinks the failure of the X-ray lies

in the fact that every point of the skull cannot be subjected to the maximum intensity of the ray.

Laryngitis.

Dr. E. B. Cayce presented Mr. H. F. B., aet 50, who first consulted him November 20, 1921, complaining of hoarseness, gradually increasing for the past four or five months. The larynx at that time showed posterior portion of arytenoids swollen and interarytenoid fold pale and congested. Left vocal cord showed two nodes, with tendency of right cord to overlap in phonation. Tuberculosis suspected. Sputum examination by Dr. Clinton E. Brush, negative. Patient went to Washington, D. C., where he delivered several addresses, after which throat cleared up entirely until recently. Several weeks ago same type of symptoms recurred. Wassermann negative.

Dr. Cayce said that the larynx was so suggestive of tuberculosis that he was very much surprised at receiving a negative report on the sputum. While he does not believe that a positive diagnosis can ever be made from mere inspection of the larynx, yet there are several elements in this case which are very suggestive of tuberculosis—i. e., the linea alba of the hard and soft palate, extending down to the tip of the uvula, this condition being regarded by Dr. J. A. Witherspoon as especially suggestive of tuberculosis; the interarytenoid condition, the arytenoids being swollen and rather faceted, and the fact that the arytenoid fold is not obliterated during phonation.

Dr. Cayce asked for a diagnosis in this case.

DISCUSSION.

Dr. W. G. Kennon said that while he had never seen a case exactly similar to this, yet to him it did not present the appearance of tuberculosis, the condition being one of edema rather than infiltration. He suggested that it might have been caused by the excessive use of the voice while suffering from laryngitis.

Dr. Herschel Ezell also doubted that the condition was tuberculosis, but found nothing upon which to base a definite diagnosis. In the absence of positive evidence, he advised antiluetic treatment, for a negative Wassermann does not disprove lues.

Dr. Cayce, replying to Dr. Ezell, said that in the presence of a negative Wassermann and the history and findings failing to indicate luetic trouble, he saw no reason why anti-luetic treatment should prove beneficial.

Dr. M. M. Cullom said that his observation had been that tuberculous laryngitis does not come on with the acuteness with which this condition developed; primary laryngeal tuberculosis being almost unknown. Under these circumstances, and in the absence of any general symptoms of tuberculosis, he would hesitate to make a diagnosis of tuberculous laryngitis.

Dr. Hilliard Wood pointed out that tuberculous laryngitis seldom if ever occurs except in fairly well developed pulmonary tuberculosis. He does not recall that in his experience he has ever failed to find pulmonary tuberculosis where he had a case of tuberculous laryngitis. He suggested that this condition might be one of malignancy, the age of the patient rendering it entirely possible. He agreed with Dr. Ezell's suggestion that antiluetic treatment be instituted, as occasionally this works miracles, even in the presence of a negative Wassermann.

Radical Mastoid for Chronic Suppurative Otitis Media With Cholesteatoma.

Dr. Fred E. Hasty presented Mr. A. J., aet 25, who, for the past 17 years has had frequently recurring pain and discharge from each ear, following some unknown type of fever. Some ten years ago had tonsillectomy, without any improvement in the ear condition. Seven years ago had tonsillectomy, following which the ears cleared up for two or three years. During an attack of influenza three years ago both ears began discharging, the left somewhat more profusely than the right. Pain more or less constant on left side of head, more especially in the mastoid region. Several otologists were consulted; all recognized methods of treatment were tried, but with little or no success.

Patient first seen by Dr. Hasty in fall of 1920. Upon examination right ear showed profuse offensive discharge; membrana tympani thickened, red, with a perforation in the posterior inferior quadrant. Left ear showed profuse offensive

discharge; membrana tympani practically destroyed; considerable granulation tissue and polypoid degeneration. Functional examination:

Right		Left
6″	Whisper	....
8′—10′	Conversation	....
....	Shout	1′ (?)
128	Lower tone limit	1024
2048+	Upper tone limit	2048
Increased	B. C.	Markedly increased
Weber to the left		

X-ray showed only sclerosis in both mastoids. Advised to have radical mastoid left ear. Patient was not seen again until Nov. 1, 1921, when the ears were found to be in the same condition as one year previous. Radical mastoid advised and done on left side, with following findings: Sclerosed cortex and cells down to the region of the labyrinthine capsule, where a cholesteatoma extending from the antrum posteriorly to the sinus wall and upwards to the fornix of the inner table of the middle fossa; ossicles, excepting the stapes, were practically destroyed. Postoperative history uneventful.

This case was demonstrated to present some of the problems of radical mastoid surgery, as follows:

1. Discharge from both ears.
2. Left ear practically deaf.
3. What will happen to the right ear if we do, or do not, operate on the left.
4. Extent of pathology in left ear. X-ray was not helpful.
5. Cosmetic results of operation.
6. Patient no longer has headache; has gained about five pounds in weight.

DISCUSSION.

Dr. Hilliard Wood congratulated Dr. Hasty upon the beautiful result obtained in this case. He inquired as to the size of the cholesteatoma.

Dr. Herschel Ezell was impressed with the fact that there was so little deformity resulting from the operation, and asked the secret of success in this respect.

Dr. Hasty, replying to Dr. Wood, said that the cholesteatoma extended from the antrum back to the sinus wall and

upward towards the middle fossa. This was all cleaned out up to the smooth cortex on the floor of the middle fossa. In answer to Dr. Ezell's question, he said that the character of the flaps, both the internal and the plastic flaps, was chiefly responsible for the lack of deformity. He described in detail the Richards method by which this operation was done. He also emphasized the fact that the X-ray failed to show this cholesteatoma. In his opinion, in these old chronic cases the X-ray shows nothing except the anatomy.

New Instruments.

Dr. Fred E. Hasty exhibited two new instruments as follows:

ANTRUM IRRIGATOR.

The object of this instrument is to combine a probe and small antrum irrigator. I find it to be satisfactory in washing any antrum, but especially in those cases where the opening is small.

(D) A solid round tip.

(E) Opening on side instead of end.

SUCTION ATTACHMENT ON TONGUE DEPRESSOR.

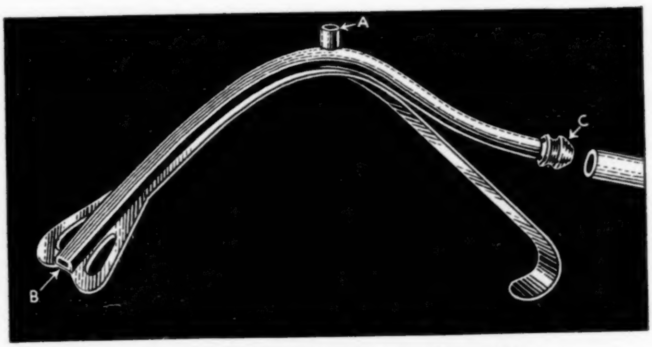
Since so much attention is being given to lung abscess following tonsillectomy, it behooves one to take every possible precaution in dealing with the blood, pus and secretions in the mouth during the operation.

I have devised this suction attachment, which may be made on most any ordinary tongue depressor. Since this drawing was made I have had a tongue depressor made by shaping two pieces of metal together and leaving a groove between to take the place of the superimposed tube. This instrument has proven very convenient in my hands and does away with the necessity of the ordinary suction tube, and in that way it almost lends another hand to the operator.

(A) By covering this opening with the thumb, suction is produced at B on the end of the tongue depressor.

(C) For connection with rubber tube on any ordinary suction machine.

Antrum Irrigator.



Suction Attachment on Tongue Depressor.